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VITAL STATISTICS

Ever since the birth of the Dionne quintuplets, there has been a very noticeable interest in blessed events among animals, that were believed to be in any way out of the ordinary. Hardly a month has passed during the last year that has not been marked by a newspaper dispatch reporting something that was believed to be "unusual" in the field of animal accouchements.

In February, a ewe at the Chicago stockyards received a great deal of attention, in the newspapers and otherwise, when she gave birth to quadruplet lambs. About a month later, a ewe on a farm near Battle Creek, Mich., gave birth to quintuplet lambs, her second set of "quints" in less than a year.

The same day that the quintuplet lambs were reported from Battle Creek, a United Press dispatch from Breslau, Germany, announced that a cow had given birth to quadruplet calves on a farm near Schlaupshof. The dispatch indicated the possibility that the mother might be bought for the government and honored as a "state breeder."

Two days later, a United Press dispatch from Miami, Florida, stated that veterinarians there were puzzled over a Maltese cat that had given birth to a litter of "quins"—three kittens and

two puppies. One Miami veterinarian is reported to have given it as his opinion that the occurrence was "very irregular." The "kittens" are females and the "puppies" are males, according to the dispatch. We believe that Bob Ripley ought to investigate this one.

Not to be outdone by any ordinary critter, Moozie, a Toggenburg goat, the property of a New York citizen, gave birth to quadruplet kids the latter part of March. A newspaper report stated that a photographer dropped in to get a picture of the happy family, and even though he succeeded in getting a photograph, he lost a camera case and the cuffs of his trousers.

A few days later, the *American Weekly* carried a feature article, telling of an Airedale, owned by the proprietor of a tourist camp near Miami, that surprised her master by delivering a litter of 14 healthy pups. Both the mother and father are said to be pure-bred Airedales, and, although larger litters have been reported from time to time, it must be admitted that a litter of 14 healthy pups is worthy of mention.

One fact that has been brought out by these different occurrences and the publicity incidental to them is that there is an acute shortage of vital statistics as far as animals are concerned. Numerous inquiries have been received at the A. V. M. A. office, from persons who wanted to know how frequently mares give birth to triplets, or cows to quadruplets, or ewes to quintuplets, as the case might be. Our literature has very little to say on the subject, and probably for a very good reason—it has never been considered worth while to record and compile such data, or even statistics on normal births. Without figures on normal births, whatever these may be in multiparous animals, it is impossible to calculate the frequency of anything abnormal.

EXHIBITS

The state associations throughout the country are being asked to coöperate with the A. V. M. A. in financing several educational exhibits that will be more or less permanent in character. It has been suggested that at least two exhibits be prepared with a view to having them shown at the New York World's Fair and the San Francisco Exposition in 1939. There will be quite a heavy expense in connection with preparing, shipping, installing, maintaining and dismantling the exhibits, and in order to be able to finance the undertaking properly the help of the state associations is absolutely essential. After the exhibits are shown

in 1939, it is planned to have them available, either entire or as units, for other purposes, such as state fairs, county fairs, live stock shows, and similar events. To date eight associations have voted contributions to the fund as follows:

Ohio State Veterinary Medical Association	$300.00
Minnesota State Veterinary Medical Society	100.00
Massachusetts Veterinary Association	75.00
Missouri Veterinary Medical Association	50.00
Nevada State Veterinary Association	25.00
North Carolina State Veterinary Medical Association	25.00
Tennessee Veterinary Medical Association	20.00
Texas, State Veterinary Medical Association of	10.00

The secretaries of several associations have inquired how much it would be in order for their organizations to contribute. This has not been an easy question to answer. The state associations are not being assessed but are being asked to give whatever they can afford. Naturally the larger bodies will be in a position to give more than the smaller ones. Purely as a suggestion for a basis on which to contribute, in cases where our advice has been asked, we have said that one dollar per member would not be far out of the way. However, this is something that each group must decide for itself.

MEMBERSHIP DIRECTORY

A new edition of the A. V. M. A. membership directory is now in the course of preparation. The new edition will be the first one to be published in about five years, there having been none since the one prepared in the spring of 1932. It goes without saying that a new membership directory has been needed badly for some time and, judging from the inquiries which have been received during recent months, the new edition will be very welcome. Naturally, in the period of five years elapsed between 1932 and 1937, there have been many changes. More than 1,000 veterinarians have joined the A. V. M. A. and the losses from various causes have been almost as great. Changes of address have been unusually numerous and, at times, have averaged as high as 75 per month over a six-month period. The new edition will be just as accurate as it is possible to make it at the time of going to press. If this notice comes to the eye of any member who has changed his address recently but who has failed to notify the Secretary's office, he should do so at once in order that he may be listed correctly in the new directory. As has been customary with previous editions, the new one will contain a complete list of the officers and committees for the current year, as well as the Constitution and By-laws.

OMAHA 1898-1937

Very few members of the A. V. M. A. realize that the meeting in Omaha in August will be the second annual convention of the national organization to be held in the Nebraska metropolis, the first having been held 39 years ago—September 6-7-8, 1898, to be exact. There were 43 members present, with 23 ladies and 49 veterinarians who were not members. The total membership was slightly over 300 at the time. One of the important business transactions was the reduction of the annual dues from $5.00 to $3.00. Another was the change of the name from United States Veterinary Medical Association to the American Veterinary Medical Association. Dr. D. E. Salmon, who was then Chief of the U. S. Bureau of Animal Industry, presided. The discussion of the report of the Committee on Diseases very clearly indicated that it was the opinion of those present that rabies and osteoporosis were the most important animal disease problems of the time. Although the intervening years have shed considerable light on both diseases, they are still with us, and rabies appears to be much more of a problem than osteoporosis right now.

PART OF OMAHA'S BUSINESS DISTRICT, AS REVEALED BY AN AIR PHOTOGRAPH. THIS IS THE HEART OF THE GATE CITY OF THE WEST, WHICH COVERS NEARLY 40 SQUARE MILES IN AREA.

EXECUTIVE BOARD ELECTIONS

Ballots have been mailed to all members in good standing in Executive Board districts 1 (Canada) and 9 (Maine, New Hampshire, Vermont, Massachusetts, Connecticut, Rhode Island and New York), containing the names of members of the A. V. M. A. nominated for district members of the Executive Board in the regular elections held every five years in these two districts.

In District 9 there was a five-cornered tie for fourth place in the primary election, which came to a close on April 20. This necessitated placing eight names on the ballot. Among the 13 candidates nominated in the two districts we find four branches of the profession represented. There are four practitioners, four sanitary control officials, three college men and two veterinarians engaged in commercial work. The list of candidates follows, with brief summaries of their A. V. M. A. activities:

DISTRICT 1

BARKER, MARK — Regina, Sask.

Veterinary Inspector, Health of Animals Branch, Canada Department of Agriculture. Graduate of Ontario Veterinary College, 1907. Joined A. V. M. A., 1913. Resident secretary for Saskatchewan, 1916-19, 1920-24, 1932—.

CAMERON, ALNE E. — Ottawa, Ont.

Chief Veterinary Inspector, Health of Animals Branch, Canada Department of Agriculture. Graduate of Ontario Veterinary College, 1908. Joined A. V. M. A., 1913. Member of Executive Board, District 1, 1932—.

CAMPBELL, JAMES A. — Toronto, Ont.

Practitioner. Graduate of Ontario Veterinary College, 1900 and 1920. Joined A. V. M. A., 1918. Member of Committee on Local Arrangements, 1910-11; fourth vice-president, 1932-33; chairman, Section on Small Animals, 1933-34; resident secretary for Ontario, 1921-23, 1935—.

HILTON, GEORGE — Ottawa, Ont.

Veterinary Director General of Canada. Graduate of Ontario Veterinary College, 1897. Joined A. V. M. A., 1911. Resident secretary for Ontario, 1915-19; member of Executive Board, District 1, 1918-32.

McINTOSH, R. A. — Guelph, Ont.

Professor of Obstetrics, Pharmacy and Therapeutics, Ontario Veterinary College. Graduate of McKillip Veterinary College, 1909. Joined A. V. M. A., 1923. Resident secretary for Ontario, 1923-32.

DISTRICT 9

EICHHORN, ADOLPH — Pearl River, N. Y.

Director, Veterinary Department, Lederle Laboratories, Inc., Pearl River, N. Y. Graduate of New York-American Veterinary College, 1900. Joined A. V. M. A., 1901. Vice-president, 1913-16, 1918-20.

HAGAN, W. A. Ithaca, N. Y.

Dean, New York State Veterinary College, Cornell University. Graduate of Kansas State College, 1915. Joined A. V. M. A., 1916. Secretary (1928-31) and chairman (1931-32) of Section on Research.

JAKEMAN, H. W. Boston, Mass.

In charge, New England Branch, Pitman-Moore Co. Graduate of University of Pennsylvania, 1909. Joined A. V. M. A., 1911. Resident secretary for Massachusetts, 1924; member of Executive Board, District 9, 1932—.

KELLY, WM. HENRY Albany, N. Y.

Practitioner. Graduate of New York College of Veterinary Surgeons, 1889. Joined A. V. M. A., 1889. Resident secretary for New York, 1898-04, 1906-10, 1919-21, 1924-25; member of Committee on Finance, 1901-02; member of Committee on Army Legislation, 1902-07; member of Committee on Necrology, 1927-28; member of Committee on Resolutions, 1933—.

MCCLELLAND, F. E. Buffalo, N. Y.

Practitioner. Graduate of Cornell University, 1909. Joined A. V. M. A., 1915. Member of Committee on Legislation, 1933—.

UDALL, D. H. Ithaca, N. Y.

Professor of Veterinary Medicine and Hygiene, New York State Veterinary College, Cornell University. Graduate of Cornell University, 1901. Joined A. V. M. A., 1913. Member (1923-27) and chairman (1927-28) of Committee on Intelligence and Education; member of Executive Board, District 9, 1930-32.

WILLS, J. G. Albany, N. Y.

Chief Veterinarian, New York Department of Agriculture and Markets. Graduate of Cornell University, 1906. Joined A. V. M. A., 1909. Member of Committee on Necrology, 1912-13; member of Committee on Resolutions, 1913-15; chairman of Section on Sanitary Science and Police, 1916-18; member of Special Committee on Bovine Tuberculosis, 1917-18; resident secretary for New York, 1933-35.

ZEPP, C. P. New York, N. Y.

Practitioner. Graduate of Cornell University, 1919. Joined A. V. M. A., 1927. Resident secretary for New York, 1935—.

Rabies Control in Chicago

To get some idea of the problem confronting the officials of large cities in efforts to control rabies, one has only to glance at the figures representing the number of ownerless dogs picked up on the streets of Chicago by the city dog catchers during the first four months of 1937. Five trucks are now in use for picking up stray dogs, from 5 a. m. to 8 p. m. each day. The personnel at the city dog pound has been increased to 34. From January 1, up to the latter part of April, 10,587 dogs were picked up and taken to the pound. During the same period, 8,527 of these dogs were destroyed. Unlicensed dogs are held for only 24 hours before they are put to death. Chicago has been under a rabies quarantine since last summer.

APPLICATIONS FOR MEMBERSHIP

(See January, 1937, JOURNAL)

FIRST LISTING

BOND, ERIC W. Central Experimental Farm, Ottawa, Ont.
B. V. Sc., Ontario Veterinary College, 1922
D. V. Sc., University of Toronto, 1935
Vouchers: C. H. Weaver and George Hilton.

COWEN, RUSSELL L. 452 Federal Bldg., Salt Lake City, Utah
D. V. M., Colorado State College, 1936
Vouchers: Jean C. Flint and I. E. Newsom.

LAMBERT, REUBEN C. c/o Montrose Beef Co., 118 N. Main St., Pittston, Pa.
D. V. M., Kansas City Veterinary College, 1917
Vouchers: A. T. Kinsley and C. E. Brown.

MALAGA ALBA, AURELIO Av. Costanera 105 Miraflores, Lima, Peru
B. Sc., M. R. C. V. S., Royal (Dick) Veterinary College, 1928
Vouchers: D. A. Tovar and J. F. Mitchell.

MCCOMB, JAMES A. 375 South St., Jamaica Plain, Boston, Mass.
D. V. M., Ohio State University, 1923
Vouchers: H. W. Jakeman and G. F. O'Malley.

PHIFER, LT. WILLIAM G. C. C. C. Co. 4407, Haughton, La.
D. V. M., Alabama Polytechnic Institute, 1936
Vouchers: Col. B. A. Seeley and J. L. Hopping.

RAMSEY, EDWARD W. Brady, Texas
D. V. M., Texas A. & M. College, 1936
Vouchers: T. O. Booth and F. C. Steinman.

ROTH, LEON 258 West 19th St., New York, N. Y.
D. V. M., New York University, 1922
Vouchers: C. G. Rohrer and J. Elliott Crawford.

WHITEHEAD, JACKSON O. 3312 Leeland Ave., Houston, Texas
D. V. M., Texas A. & M. College, 1935
Vouchers: Chas. W. Neal and Matthew E. Gleason.

WILLIAMS, HARRY U. Yerington, Nev.
D. V. M., Saint Joseph Veterinary College, 1917
Vouchers: Edward Records and L. R. Vawter.

Applications Pending

SECOND LISTING

(See April, 1937, JOURNAL)

Abrams, Guy E., Box 2522, Reno, Nev.
Altaker, Lt. Robert R., 331 Wabash Bldg., Pittsburgh, Pa.
Bagley, Russell O., 1510 Rainier Ave., Seattle, Wash.
Blackburn, Loren C., 332 Federal Bldg., Lincoln, Neb.
Brown, Roswell L., 1817 Church St., Evanston, Ill.
Cain, H. Driscoll, Box 572, New Hyde Park, L. I., N. Y.
Claus, Norman G., 3987 Maybury Grand Ave., Detroit, Mich.
Cooper, James H., Box 503, Laredo, Texas.
Fisherman, Lt. Henry, 1400 Runnels St., Big Spring, Texas.
Howarth, Carl R., 320 4th Ave. S., South Saint Paul, Minn.
Huebner, Richard A., 2723 N. 13th St., Philadelphia, Pa.
Maugel, John P., 525 Widener Pl., Philadelphia, Pa.

Meisels, Raphael, 1749 49th St., Brooklyn, N. Y.
Miller, Donald, 105 Arizona State Bldg., Phoenix, Ariz.
Mueller, Robert A., 2116 E. Colorado St., Pasadena, Calif.
Mullen, Hugh E., 330 Federal Bldg., Madison, Wis.
Newberg, Aaron, 1620 E. 35th St., Kansas City, Mo.
Riddell, William K., 1909 State St., Santa Barbara, Calif.
Smith, Rollin R., R. 2, Box 345, Chino, Calif.
Upchurch, Lt. John W., 1601 N. Ballinger St., Fort Worth, Texas.
Walker, Donald M., 4901 S. 33rd St., Omaha, Neb.

The amount which should accompany an application filed this month is $8.33, which covers membership fee and dues to January 1, 1938, including subscription to the JOURNAL.

State Officials Appointed

Dr. R. A. Hendershott (O. S. U. '17) was named chief of the Bureau of Animal Industry, New Jersey Department of Agriculture, on April 16. He has been connected with the department since 1926, as veterinary pathologist and acting chief. Dr. Hendershott held the latter position since the fall of 1936, following the death of Dr. J. H. McNeil, who had been chief since 1917. Dr. Hendershott ranked first in the promotional test given by the New Jersey Civil Service Commission.

Dr. J. L. Axby (Chi. '03) has been reappointed State Veterinarian of Indiana for a four-year term dating from May 1. He was originally appointed to this office in 1933. It was reported that there will be no changes in the personnel of the Indiana State Live Stock Sanitary Board.

Dr. Ben Anderson (Iowa '26), of Canton, S. Dak., has been appointed Director of the Division of Animal Industry of the South Dakota Department of Agriculture.

Dr. Thomas A. Elliot (Wash. '14), of Lewiston, Idaho, has been appointed Director of the State Bureau of Animal Industry.

Styles in Dogdom

As is the case with clothes, automobiles and many other things in everyday use, in which styles change from year to year, the popularity of certain breeds of dogs rises and wanes with the passage of time. Right now the cocker spaniel apparently has supplanted the Boston terrier in popularity, if the registrations with the American Kennel Club may be taken as an indication of the current trend. Of the 6,325 dogs registered during February, 1,024 were cocker spaniels, with the Boston terriers a poor second with 742 registrations, 208 less than for the corresponding month of 1936. Greyhounds also appear to be winning more public favor at present.

COMING VETERINARY MEETINGS

Connecticut Veterinary Medical Association. Dr. F. F. Bushnell's Hospital, Manchester, N. H. May 5, 1937. Dr. George E. Corwin, Secretary, State Office Bldg., Hartford, Conn.

American Animal Hospital Association. Book-Cadillac Hotel, Detroit, Mich. May 6-8, 1937. Dr. D. A. Eastman, Secretary, 901 Nineteenth St., Moline, Ill.

Idaho Veterinary Medical Association. (Joint meeting with the Inland Empire Veterinary Medical Association.) Pullman, Wash. May 8, 1937. Dr. E. M. Gildow, Secretary, University of Idaho, Moscow, Idaho.

Ak-Sar-Ben Veterinary Medical Association. Elks Building, Omaha, Neb. May 10, 1937. Dr. W. H. Riser, Secretary, Glenwood, Iowa.

Chicago Veterinary Medical Association. Anti-Cruelty Society, 157 W. Grand Ave., Chicago, Ill. May 11, 1937. Dr. O. Norling-Christensen, Secretary, 1904 W. North Ave., Chicago, Ill.

Hudson Valley Veterinary Medical Society. West Point, N. Y. May 11, 1937. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

San Diego County Veterinary Medical Association. San Diego, Calif. May 11, 1937. Dr. Donald E. Stover, Secretary, Zoölogical Research Bldg., Balboa Park, San Diego, Calif.

Willamette Valley Veterinary Medical Association. Oregon City, Ore. May 12, 1937. Dr. Elwyn W. Coon, Secretary, Forest Grove, Ore.

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. May 18, 1937. Dr. C. C. Foulk, Secretary, 1103 E. 47th St., Kansas City, Mo.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. May 19, 1937. Dr. B. B. Coale, Secretary, 203 Administration Bldg., Union Stock Yards, Los Angeles, Calif.

Colorado Veterinary Medical Association and Colorado State College Veterinary Short Course. Fort Collins, Colo. May 19-21, 1937. Dr. Jay H. Bouton, Secretary, Aurora, Colo.

Michigan-Ohio Veterinary Medical Association. Blissfield, Mich. May 21, 1937. Dr. J. C. Schwabland, Acting Secretary, Blissfield, Mich.

Keystone Veterinary Medical Association. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa. May 26, 1937. Dr. M. W. Allam, Secretary, Media, Pa.

Southern California, Veterinary Hospital Association of. Los Angeles, Calif. June 1, 1937. Dr. L. B. Wolcott, Secretary, 1434 W. Slauson Ave., Los Angeles, Calif.

New York City, Veterinary Medical Association of. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. June 2, 1937. Dr. J. B. Engle, Secretary, Box 432, Summit, N. J.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. June 2, 1937. Dr. Milton R. Fisher, Secretary, 3678 Dover Pl., Saint Louis, Mo.

Houston Veterinary Association. Houston, Texas. June 3, 1937. Dr. Claude Canion, Secretary, 409 Link Rd., Houston, Texas.

East Tennessee Veterinary Medical Society. White Surgical Supply Building, Knoxville, Tenn. June 5, 1937. Dr. Robert L. Hummer, Secretary, 312 W. Church Ave., Knoxville, Tenn.

Oklahoma Veterinary Medical Association. Skirvin Hotel, Oklahoma City, Okla. June 7-8, 1937. Dr. F. Y. S. Moore, Secretary, McAlester, Okla.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. June 9, 1937. Dr. F. D. Egan, Secretary, 17422 Woodward Ave., Detroit, Mich.

Ohio State University Veterinary Conference. Columbus, Ohio. June 16-18, 1937. Dr. Oscar V. Brumley, Dean, Ohio State University, Columbus, Ohio.

California State Veterinary Medical Association. Long Beach, Calif. June 21-23, 1937. Dr. Chas. J. Parshall, Secretary, Brentwood, Calif.

Eastern Iowa Veterinary Association, Inc. Mechanicsville, Iowa. June 22, 1937. Dr. John J. Strandberg, Secretary, 1005 Eighth Ave., Belle Plaine, Iowa.

Michigan State Veterinary Medical Association. Michigan State College, East Lansing, Mich. June 22-23, 1937. Dr. E. C. W. Schubel, Secretary, Blissfield, Mich.

Wisconsin Veterinary Medical Association. Madison, Wis. June 29-30, 1937. Dr. B. A. Beach, Secretary, University of Wisconsin, Madison, Wis.

New York State Veterinary Medical Society. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. July 7-9, 1937. Dr. F. F. Fehr, Secretary, 243 S. Elmwood Ave., Buffalo, N. Y.

American Veterinary Medical Association. Hotel Fontenelle, Omaha, Neb. August 16-20, 1937. Dr. H. Preston Hoskins, Secretary, 221 N. La Salle St., Chicago, Ill.

STATE BOARD EXAMINATIONS

Iowa Veterinary Medical Examining Board. State Capitol, Des Moines, Iowa. June 15-16, 1937. Applications must be on file at the Division of Animal Industry not later than 8:00 a.m., on June 15. Further information may be obtained from Dr. H. A. Seidell, Secretary, State House, Des Moines, Iowa.

Nebraska Bureau of Examining Boards. State House, Lincoln, Neb. June 14-15, 1937. Applications must be on file at the Bureau at least 15 days prior to date of examination. Mrs. Clark Perkins, Director, Bureau of Examining Boards, State House, Lincoln, Neb.

California Board of Examiners in Veterinary Medicine. Long Beach, Calif. June 21-23, 1937. Applications must be filed with the Secretary at least 30 days prior to date of examination. Dr. Nelson E. Clemens, Secretary, 183 Castro St., Hayward, Calif.

Illinois State Board of Veterinary Examiners. Chicago, Ill. July 19-20, 1937. Dr. L. A. Merillat, president of the Examining Committee, asks candidates to file their applications with the Director of Registration and Education, Springfield, Ill., not later than July 1.

Massachusetts Board of Registration in Veterinary Medicine. Department of Civil Service and Registration, State House, Boston, Mass. June 30-July 1, 1937. Applications may be obtained from the Secretary. Dr. E. W. Babson, Secretary, Gloucester, Mass.

Semi-Centennial for Pennsylvania Alumni

The Alumni-Day Committee of the Veterinary Alumni Society, University of Pennsylvania, wishes to repeat the invitation which appeared in the April issue of the JOURNAL. To all veterinary alumni of all the colleges and universities the nation over, and their friends who are indeed friends of the profession as a whole, it is the sincere hope of the Pennsylvania alumni that as many as possible will attend the celebration of June 4 and 5, 1937, at Philadelphia, commemorating the Society's completion of its first 50 years. Anyone wishing further particulars will be accommodated upon writing Dr. E. T. Booth, Secretary, 39th Street and Woodland Avenue, Philadelphia, Pa.

Words are but holy as the deeds they cover.—Shelley.

INFECTIOUS ENTEROHEPATITIS*

By A. J. DURANT, *Columbia, Mo.*
Department of Veterinary Science, University of Missouri

Infectious enterohepatitis of turkeys is the most serious disease confronting this industry. In Missouri it is estimated that 90 per cent of turkeys that die are affected with this disease. Apparently it exists everywhere turkeys are raised to any extent and it is probably found in every state in the Union.

The cause of the disease is a protozoan organism which has been designated by Tyzzer[1] as *Histomonas meleagridis* and it is probable that this is the correct classification of this organism. Tyzzer believes it to be an organism having both ameboidal and flagellate characteristics and has ignored the classifications of previous workers.

According to Tyzzer[2] this organism has three distinct phases in its life cycle; invasive, vegetative and resistant phases. In the invasive phase the organism is distinctly amebiform and varies considerably in size. It measures, when rounded, from 8 to 17 microns in diameter in fixed tissue. Long forms may measure 30 microns in length. These forms are found in all the tissues involved in this disease.

In the vegetative phase the organism is of large size—from 21 x 15 to 12.5 x 12 microns. It accumulates in great numbers and distends the host tissue which have not yet reacted to its presence. Many of these forms survive to pass into the resistant stage.

The resistant phase is characterized by the development of a dense surface membrane, by an increase in the amount of coagulable material, and probably by loss of water on the part of the cytoplasm. These forms are comparatively small, slightly less than 5 microns in diameter.

Multiplication of *H. meleagridis* in the tissues is by binary division, which resembles that of the trichomonads. This division is most active in the invasive phase, continues in the vegetative, and ceases in the resistant phase.

The organism is easily killed and cannot live outside the host body alone and only in ideal media can it exist for 80 days. This suggested to Tyzzer that there was possibly an intermediate host connected with it in its life cycle. Experiments have shown that the ovum of *Heterakis gallinae* is an important source of the

*Contribution from the Department of Veterinary Science, Missouri Agricultural Experiment Station. Journal Series No. 499. Presented at the seventy-third annual meeting of the American Veterinary Medical Association, Columbus, Ohio, August 11-14, 1936.

virus in the natural transmission of blackhead in turkeys, and four investigators[3, 4] have established definitely that the cecal worm serves as an invertebrate host of *H. meleagridis*. It is now well known that turkeys seldom contract blackhead in the absence of this parasite of the ceca.

Tyzzer[5] has succeeded in demonstrating the organism *H. meleagridis* in half-grown cecal worms from cases of blackhead. No one, however, has yet demonstrated the organism of enterohepatitis in the cecal worm eggs themselves.

The cecal worm serves as an admirable intermediate host for *H. meleagridis*. The embryonic worm penetrates into the mucosa of the ceca in the early part of its cycle in these organs, producing injury to a susceptible tissue and at the same time carrying the protozoan organism to the injured site, thereby assuring the host of infection with the organism causing enterohepatitis. Just how this transfer takes place is not known.

Because of the close relation between the cecal worm and *H. meleagridis*, any program for the control of enterohepatitis should concern itself with both of these disease-producing entities.

Under natural conditions organs affected by blackhead are the cecal pouches and the liver. Experimentally we have produced blackhead in practically all organs of the body by feeding massive doses of ground-up diseased liver, while it was still warm, after removal from the body of a bird affected with the disease.

The disease in a natural outbreak first invades the blind portion of the cecum. The organism penetrates the tissue and multiplies very rapidly. A few days after invasion of the cecal wall it metastasizes through the blood-stream to the liver, where it produces necrosis and typical circumscribed abscesses.

Proof that the organism under natural conditions first invades the ceca can be shown by abligating[6] some birds; that is, separating the two ceca from the main gut, then exposing the birds to infected grounds. Only a small percentage of abligated birds under these conditions will develop blackhead disease, whereas, unabligated birds running on the same infected grounds will all die.

SYMPTOMS

The disease may appear as early as three weeks on badly infected ground, though usually the birds do not contract the disease that early, but it should be remembered that turkeys of all ages are susceptible to blackhead, provided they have not been exposed to the disease previously. Even adult birds, when placed on infected grounds, will die in large numbers if they

have not previously been exposed to the disease, or abligated.[6] The greatest losses in Missouri occur in poults from one to five months of age.

In general, the older the birds are the more resistant they are and fewer will actually die with the disease when exposed to infected grounds. As in most diseases, a few turkeys seem to be

FIG. 1. A young turkey tom in the last stages of blackhead disease. One can visualize the condition of the liver of this turkey by examining figure 2.

naturally resistant to blackhead or else acquire resistance by having a slight attack of the disease. It is probable that the few birds which are observed on the ordinary farm running with chickens are of this nature.

In young turkeys the first thing noticed at the onset of the disease in the flock will be a few birds which will lag behind the rest of the flock. If the birds are very young, the poults will die without showing any marked symptoms except weakness, droop-

ing wings, and lagging behind the flock. It is not uncommon to have young poults die in convulsions with their heads drawn back or twisted to one side. The older birds (fig. 1) are much more likely to show the characteristic orange-yellow droppings of the disease.

Diseased birds in a flock may be detected by examination of the droppings. This is a practical and simple way of determining which birds have the disease before they have shown symptoms of sickness and it will be possible to isolate the sick birds earlier. If the characteristic orange-yellow droppings are not in evidence, an examination of a dead or dying bird will confirm the diagnosis.

Pathology of the Disease

The disease produces typical ulcers of the ceca and abscesses of the liver. It affects either one cecum or both of these organs. As has already been mentioned, the ulcers usually occur towards the blind end or apex of the cecum and vary in size from small, dirty-colored, yellowish ulcers to very large ulcers which may be several inches in diameter. These lesions penetrate deeply into the mucosa and muscular tissues of the cecum and it is not uncommon for them to produce such extensive destruction of the muscular wall as to cause a perforation which results in peritonitis and death within a short time.

The change produced in the liver is enlargement of the organ accompanied by the formation of abscesses throughout. These abscesses vary greatly in size from small necrotic foci to large abscesses an inch in diameter. The lesions vary in color from almost white to a greenish yellow and brown. The abscesses on the surface are concentric and sunken, which will enable one to distinguish them from tuberculosis lesions. The extent of the disease in the liver varies from just a few large abscesses with a small amount of enlargement of the liver to cases where the liver is greatly enlarged and almost a solid mass of abscesses (fig. 2).

Mortality

The death-rate in this disease is high and in young poults, four to five months old, 90 to 95 per cent of the birds will die with the disease when running on infected ground. In older turkeys the losses are not so great, although the mortality may extend over a considerable period of time. The fact that not all turkeys develop the disease at the same time has great advantage in controlling an outbreak in a flock, since practically there never is a time in the course of an outbreak when some of

the birds may not be salvaged if proper control measures are instituted.

Control Measures

The nature of enterohepatitis does not render it amenable to medicine or drugs. Prevention is the only method by which the disease may be controlled and prevented. Sanitation is the main feature in the control of blackhead in turkeys.

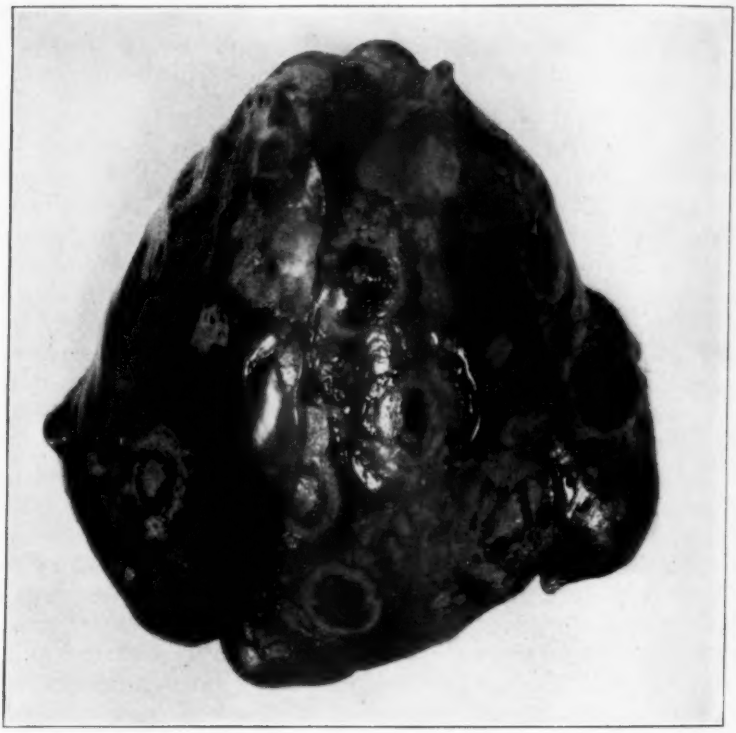

Fig. 2. Liver of a turkey badly affected with blackhead. Very little healthy tissue remains in this organ as a result of invasion of *Histomonas meleagridis*.

Two systems are now in use. One is to raise the birds in confinement on raised wire floors and the other is to raise them on clean grounds and to rotate the grounds frequently. The most successful method, where the ranges are limited, is to have four separate ranges upon which to rotate the turkeys during the year.

In any case it is highly important that young turkeys be kept away from older birds and that both be kept entirely isolated

from chickens or where chickens have been within the last two years.

A modified plan which is quite successful is to raise the poults on wire runs until they reach the age of from eight to twelve weeks and then put them out on alfalfa ranges in which the four areas mentioned can be utilized for rotating the birds.

Another method of prophylaxis which at the time appeared to have considerable practical value was cecal abligation. This is a surgical operation which was developed by the author and consisted in ligating the two ceca which resulted in a permanent separation of these organs from the main gut. Though the original experiments demonstrated very clearly that an abligated bird would not develop blackhead when running on infected grounds, the birds used in these experiments were almost grown and were naturally more resistant to blackhead. Later experiments showed that a small percentage of turkeys developed blackhead even when abligated. This fact, however, did not mitigate against the operation. The drawback to the practical application of cecal abligation was, first, the high mortality from the operation as performed by the author; second, the separated ceca in from seven to 32 months became greatly enlarged, so that unless they were removed from the body of the bird they eventually would cause death.

Schlotthauer, Essex and Mann[7] greatly improved the technic of this operation so that the mortality was reduced to 1 per cent or less. They, however, do not regard the operation as practical for the control of blackhead. They found in their young poults there was considerable mortality even in abligated birds. There might be, however, special conditions under which this operation could be utilized for the control of enterohepatitis in turkeys. The author would recommend to those interested the technic developed by Schlotthauer, Essex and Mann.

Control Measures in an Infected Flock

It has already been mentioned that all turkeys in a flock do not develop the disease at the same time and that many birds may be salvaged from an infected flock if proper control measures are instituted.

If the disease appears in a flock at any time it means but one thing—that the grounds where the birds are located contain infection and that it will be necessary to move the flock to clean grounds in order to check further spread. Preliminary to this removal, the birds should be carefully culled and all visibly sick fowls removed from the healthy flock. In selecting infected birds

advantage should be taken of the knowledge that many, especially large turkeys, will show the orange-yellow droppings that are characteristic of the disease before the birds have shown any signs of being sick.

After the apparently healthy flock has been carefully culled, it is then advisable to give all the birds in the flock a comparatively large dose of turpentine in capsules. The dose is about 2 cc for an adult bird. This is to kill the protozoan organisms that are still free in the digestive tract and have not yet attacked the tissues of these organs. In this way, theoretically, many birds will be prevented from developing blackhead. After the administration of the turpentine, the birds can then be put on clean grounds.

These recommendations apply to small flock-owners who constitute an important part of the turkey industry of the United States and the recommendations are practical for small flocks of turkeys and will, when properly applied, result in an enormous saving financially to the turkey-owners of the Central West.

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Discussion

Dr. E. P. Johnson: Dr. Durant, I would like to ask if you think the cecal worm is an essential host for the blackhead organism? I understand that blackhead can be produced by injection and not necessarily by feeding.

Dr. Durant: My answer is that it is not an essential host. It is one of the essentials, I should say, because if you abligate a bird and remove the site for the cecal worm, presumably it cannot exist in the digestive tract; and in those cases, I would say a large percentage of those birds—perhaps two-thirds—would not develop blackhead, even when running on infected ground. Therefore, it certainly has an important place in the picture; but the fact that turkeys get it after they are abligated, and that you can inject it into them and produce the condition, seems to prove that it is not an essential.

With respect to using ipecac in cases of enterohepatitis, I might say we treated 75 birds afflicted with typical enterohepatitis and 62 per cent of them recovered. Please notice how I state that: I say that 62 per cent of them recovered on ipecac.

We thought last year that we had discovered a cure for blackhead. We treated 16 birds and twelve of them recovered. However, we decided we had better be on the safe side, so we began treating every other bird with the drug, leaving every other one untreated, and when we finished that experiment we found there was no difference in the number of recoveries at all. (Laughter)

We were going to revolutionize the treatment of enterohepatitis with that drug. It was atabrine, a drug used for malaria. I came from the South and would naturally choose some southern drug. However, the medical profession suggested it to me as a possible amebicide, but it did not work.

DR. C. D. CARPENTER: Where you abligate the pouch and leave a small stub, do you find that cecal worms can accommodate themselves to the new, small cul-de-sac which is left there?

DR. DURANT: I have never seen a cecal worm, but I have witnessed a number of cases of blackhead in the stump. In one case involving a typical ulcer in the stump, there were about a dozen tiny rocks present.

DR. CARPENTER: Which may have injured the mucosa?

DR. DURANT: That is what we thought. In the other stump, there were no rocks or lesions.

Speaking of treatments, has anybody tried the well-known colloidal iodine?

DR. E. C. W. SCHUBEL: I rather hesitate to mention the fact, but I have been using it for three or four years.

I am in a section of the country where the farmers have small flocks. I do this as a matter of service to my clients. They may have 20, 30, 40 or perhaps 75 turkeys per flock.

I count the treatment of turkeys as part of my income each fall. There will be hardly a day, from now on, that I will not get a call to treat some turkeys.

On Monday, just before noon, a lady came to the office with one of her turkeys which was dead. I found cecal worms present, just as you described. The ceca were about six or seven inches long, and enlarged considerably. The bird had the same kind of liver that you described.

I said to the lady, "The only thing we can do is treat your turkeys with colloidal iodine, and if you will bring them to the office we will do it." That is a service we render every day.

Last year, I began treating a flock of 120 in the latter part of July, giving them one-half ounce of the colloidal iodine. I said to the owner: "In about six weeks, we will treat these again and at that time we will vaccinate against fowl-pox." He replied that he wanted me to come out to treat them. We treated them again and they went through the rest of the season all right. About the first of November, because the owner feared that something might develop, he asked us to come out and treat them again. We went out there, and by that time we had to use the 24-inch dosing apparatus on the turkeys because they were fully grown. That man does not attempt to raise turkeys without having them treated each year. They have been treated once this year. That is the way we render this service.

On sick birds, we have a small percentage of recoveries. I checked with this particular man, whom I began working for last year. Of five sick birds which he presented to me, three made recoveries and grew to adult turkeys.

I feel this is a valuable service for those veterinarians who want to build up a poultry practice. It is a lot of work to stand in one place and put this dosing catheter down the necks of turkeys for one hour, two hours or three hours at a time, using a two-ounce bulb, but it is a service which we can give our clients to keep out the county agents. That is what started it in my community. The county agent

began it, or perhaps it was the service man from some feed company. However, by doing this ourselves, we have these agencies removed from the scene. They do not dare compete with us on that service. We are aiming to get it for the veterinarian.

However, I find some veterinarians do not want to do it. That is the reason I urge them to do such things as treating turkeys, because I believe it will be found in most instances that veterinarians serving the small flock-owners give their clients better service with colloidal iodine than anything which has been attempted. I have been doing it for three or four years. I know there are a number of men here who have had some experience with it. For that reason I hesitated to say anything because I was afraid you would say, "Well, what are you trying to do—sell somebody's product?" That is the feeling among some veterinarians—that you are trying to promote somebody's product; but it is not that motive which urges me to speak on this subject, because I believe it is a service which can be given to these owners of small flocks.

DR. CARPENTER: I would like to ask what fee you get for that type of work?

DR. SCHUBEL: If the owners bring the birds to me, I generally charge ten cents a bird.

DR. CARPENTER: Including the cost of the iodine?

DR. SCHUBEL: I buy it for $2.45 a pint, to which I add seven pints of water, making eight pints in all. For a small turkey, the dose is one-half ounce; for a turkey the size of a hen, one ounce; for the adult turkey, the dose is two ounces. The mortality has been very small. There has been only one instance when in a group of turkeys some were killed. That was due to an accident, so it did not involve the use of colloidal iodine.

DR. JOHNSON: Do you recommend rotation of yards after treatment?

DR. SCHUBEL: Yes, I do; but it is very difficult to make your clients understand. They have not learned the basic fundamentals. In the first place, some of them have no business raising turkeys because they are not equipped for it, especially those people who are situated as they are on a great many farms in southern Michigan and northern Ohio. If turkey farms are located on higher land, where you have good drainage, they are all right. However, it is our job to take care of their flocks.

DR. DURANT: I think the answer to the iodine treatment is that if it gets rid of the cecal worms it certainly would help prevent blackhead. I do not think there is any question about it, because we know that the parasite is the thing which incites blackhead in most of the cases.

DR. HUGH HURST: I would like to ask a question about this treatment of 2 cc of turpentine which Dr. Durant spoke of as a means of ridding them of the roundworms. At what age would you give that?

DR. DURANT: That is a dose for an adult turkey, and you have to reduce it according to the age. At any time you have an outbreak, you administer the turpentine and move them to clean grounds.

DR. HURST: I might state for your information that I have in my care approximately a million turkeys. If we began dosing them individually it would be a rather laborious task. I am wondering if there is some other means which we can employ and which would be effective.

DR. DURANT: Dr. Frederick, do you want to tell them how you administer the turpentine?

DR. L. D. FREDERICK: Dr. Durant and I have been coöperating on the experimental use of turpentine as a preventive of blackhead in turkeys. For four-week-old birds, I use 0.25 cc; for a bird beyond that age, correspondingly larger amounts. When blackhead breaks out in a flock, turpentine is administered every other day for a period of

about four doses; then we give it every third day throughout the season. On older birds, we gradually increase the dose until at an age of from 14 to 16 weeks, we give 1.5 cc.

I might explain how we administer this treatment. We feed wet mash twice a day to our birds. We take the ordinary dry mash and wet it, either with water or buttermilk, whichever we happen to be using at that particular point. For the use of turpentine we figure out the amount needed in accordance with the number of birds we are handling. We mask the turpentine so the birds will take it by adding one ounce of oil of sweet almond to the gallon of turpentine. In case we do not want the feeder to know what we are using, we add one ounce of pulverized alkanet root. This gives a nice, cherry-red color; and by masking the turpentine with the oil of sweet almond which gives an almond odor, we get the turkeys to eat the mash with the turpentine in it quite readily.

DR. CARPENTER: Do they eat that mash readily?

DR. FREDERICK: Yes. Of course, whenever you change the feed on birds you have to do it gradually. You have to teach them to eat the new feed. I am speaking of wet mash now. If they are used to eating dry mash and you change to wet mash, and you put too much in the feed-troughs, they are going to leave some and you will have some sour feed.

DR. CARPENTER: Do you return to dry mash when you are not treating them, or do you feed them the wet mash all the way?

DR. FREDERICK: We feed them the wet mash morning and evening, all the way, and medicated mash once every third day. Dry mash also is supplied in separate feeders.

We do not care to report our conclusions on this matter at this time, but we have experimented with it for quite some time. We have quite an extensive experiment going on right now in Michigan, using 5,000 birds on a split basis, 2,500 in each lot. We have another group in northern Iowa, where the test is not controlled, and where we are going to use it for the entire period. We are not as yet ready to report beneficial results or otherwise, as the data are too limited to date.

I would like to report, however, that after four treatments, using turpentine, I have yet to find any intestinal parasites. Of course, before the time our birds go on the range, they are brooded in brooder-houses, either on wire, or on some sort of litter which is highly absorbent and which is changed biweekly. Then at eight to ten weeks, sometimes earlier, they are taken out on the range. After they go on the range, they are moved to a new location every week. This helps us very materially in controlling disease and parasitic infestation.

In connection with that point, I would like to ask a question, if I may. In this rotation of grounds, where we are having disease—especially blackhead—how long would it suffice to leave the ground vacant of turkeys before we could move them back on it again?

DR. DURANT: I think the answer to that would depend on whether you are using four plots in the course of a year.

DR. FREDERICK: We are not restricted to plots. We rent enough ground to move every week throughout the entire season. We market our turkeys at about 24 weeks of age.

DR. DURANT: I think in figuring the length of time that blackhead will exist on the grounds you have to consider the parasite. The cecal worm is the agent which is responsible. Presumably, the disease does not exist outside unless the cecal worm is found there; and two years certainly would be required to get rid of the cecal worm.

I would like to make myself clear with regard to the use of turpentine. I do not wish to mislead anyone. The idea of this turpentine in the digestive tract is not primarily to rid the turkey of the cecal

worm, although it may do that. The purpose in putting turpentine in the digestive tract was to destroy the parasite or organism which caused blackhead and which was free in the digestive tract. If this turpentine—though we do not regard it as a good worm remedy—will get rid of the cecal worm, it is serving a double purpose, and that is better than we expected.

If the colloidal iodine will remove the cecal worm, I think it will be very valuable as a means of preventing blackhead.

DR. JOHNSON: Before we get too enthusiastic over the treatment of blackhead disease with any remedy, I just want to say one thing about some experiments which I carried out with colloidal iodine, and where I controlled the experiment, giving iodine to half of the turkeys and allowing the remainder to go without it. In conducting those experiments, we found that the birds which received the iodine developed blackhead just as readily as the ones which did not get it.

With the birds which had blackhead disease, we treated them with iodine and every one of them died; we could not cure a single one. Personally, I cannot conceive of any remedy which is going to restore the tissue which is destroyed, as it is in the liver with blackhead disease.

I would like to know what the therapeutic action is with relation to the thing they are working with; just how does the colloidal iodine restore this tissue? What does it do? Does it kill the organism? How does it reach the liver? Just how does it restore the tissue? That is the thing I am interested in.

The same is true with the turpentine treatment. If it will destroy the worm, all well and good, but I think we have to rely on sanitation.

DR. THOMPSON: Dr. Durant, you mentioned the inability of this organism to exist very long outside of the intestinal tract. I was wondering if, after the outbreak is established in a flock, it would not exist long enough to perpetuate the disease when the organism has contaminated the drinking water and feed? Would it not always be necessary to have a supply of cecal worms or cecal worm eggs coming along to perpetuate a disease in a flock over one season?

DR. DURANT: The question is: Would the droppings of a bird infect another bird directly? If one bird picked up the droppings of another while still warm, I would say that it would become infected very quickly and there would be no need for any intermediate, invertebrate host, which we have already mentioned, such as the cecal worm. There may be other kinds of hosts which carry this parasite. We do not know all of them, but certainly if a turkey picks up the warm droppings of another bird, which is infected, I believe he would develop the disease very quickly. If it remained on the ground for an hour or more, the disease would probably not be transmitted.

DR. FREDERICK: I believe that is very true, basing it on the experience I have had. I believe it reverts back to Dr. Johnson's idea of very strict sanitary measures in controlling this condition, especially in large flocks.

DR. HURST: I am interested in knowing, also, if there has been any experimental work done with any form of nicotine in controlling these cecal worms, which will accomplish the same thing the turpentine is reported to be doing.

DR. DURANT: We are carrying on experiments now with respect to that, feeding large amounts of tobacco dust in the mash. We have a group of birds which are receiving ordinary mash, and another group which receive mash containing the tobacco dust, nicotine sulfate, 4 per cent.

The whole trouble in Missouri this summer has been that it has been so dusty that we have not had the sick control birds we should

have over the birds which were getting the preventive treatment, or the others, and consequently we have not been able to collect the necessary data. Ordinarily, if you put a flock of poults on infected grounds, you can expect to have birds infected with the disease in 14 days, regularly, and those poults will all be dead—or practically all of them—within three or four weeks after the disease appears. However, this summer it has been so dry, and conditions have been so unfavorable to the appearance of blackhead, that we have not had it among the flocks.

DR. L. M. RODERICK: With reference to the powdered tobacco, some work was done at our laboratory some years ago in an attempt to control blackhead by its use, and it was found ineffective. We also used it in the mash in our experiments.

DR. DURANT: What percentage did you use? Do you recall the figure?

DR. RODERICK: I cannot say definitely, but we were using it in varying proportions, and the mash was pretty heavily loaded. I think the figure was up to 6 per cent; that is just my recollection.

DR. F. R. BEAUDETTE: Did I understand you to credit Tyzzer with establishing the fact that the embryonic cecal worm is the intermediate host of the blackhead parasite?

DR. DURANT: No. I said he had demonstrated the parasite in the cecal worm after it was hatched, but he had never shown it in the egg. He made histological sections showing the parasite in the cecal worm just after it was hatched.

Retrospection

Dr. T. P. White, who is now stationed in London, England, as the representative of the U. S. Department of Agriculture, has written a history of the Bureau of Animal Industry in blank verse under the title, "Retrospection." In this unusual story Dr. White recounts the triumphs of veterinary science in the United States over such foes as contagious pleuro-pneumonia, foot-and-mouth disease, dourine, Texas fever and hog cholera. The campaigns against scabies, tuberculosis, Bang's disease and lesser foes are depicted, as well as the development of the U. S. system of meat inspection. The history is concluded with an appreciative tribute to the trio of great veterinarians who have headed the Bureau during its more than half-century of service—Salmon, Melvin and Mohler.

A small edition of "Retrospection" has been printed and anyone wishing to secure a copy may order through the JOURNAL office. The price is 30 cents per copy.

Brought Home the Wallet

An unusual story of a police dog comes from an East Orange, N. J., resident. The dog carried home a wallet containing $45.00, which had been lost by the driver of a moving van.

TRICHOMONIASIS OR TRICHOMONAD ABORTION IN CATTLE*

By H. SCHMIDT, *College Station, Texas*
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European literature credits Mazzanti[1] as being the first observer to have discovered trichomonads in cattle (three cases). He described the organism rather inadequately under the name *Trichomonas utero-vaginalis vitulae.* Twenty-five years later, Hopfengärtner[2] found trichomonads in the stomach fluids of a 7-month-old bovine fetus and, two years later, Pfenniger[3] reported a similar finding. In 1928, Riedmüller[4] published rather comprehensively on his findings of trichomonads in cattle. He recognized them as a causative agent of abortion and classified it as of an infectious nature. He described the organism very carefully as *Trichomonas foetus,* but he later suggested a change to *T. bovis.*[5] By this description he placed the recognition of the organism on a sound basis, although Wenrich and Emmerson[6] added minor details to the morphology of the organism and renamed it *Tritrichomonas foetus* (Riedmüller). They rejected Mazzanti's name because it is trinomial and also Riedmüller's suggested change to *T. bovis* because such a change would be in violation of rules of nomenclature. Witte[7, 16] speaks of *T. genitalis* which is also accepted by Küst.[27] Gehring and Murray[8] accept Riedmüller's second suggested designation, *T. bovis,* and again describe it.

The body of the parasite is spindle-shaped, varies from 10 to 25 microns in length and is usually one-third to two-fifths as wide as long. It possesses three anterior flagella which are somewhat unequal in length among themselves and are about as long or longer than the body. The posterior flagellum is the prolongation of the marginal filament of the undulating membrane and trails posteriorly as a free flagellum. It is about as long as the anterior flagella. The undulating membrane is placed dorsally, runs nearly full length of the body, follows a spiral course and has four or six undulations. An accessory filament lies close to the marginal filament, but terminates at the posterior end of the costa. It has a thick axostyle extending axially from the blepharoplast where it begins with a club-shaped expansion and emerges posteriorly from the body where it tapers to a sharp point which may be prolonged into a slender spine. The nucleus lies normally within the anterior third of the body and dorsal

*Presented at the seventy-third annual meeting of the American Veterinary Medical Association, Columbus, Ohio, August 11-14, 1936.

to the axostyle. It is oval in shape, from 3.5 to 5 microns long and about half as wide.

In fresh material under the high dry power the organism can readily be seen in active motion, rotating comparatively rapidly counter-clockwise and moving slowly from place to place by jerky movements along an irregular path. The flagella can be seen to be rapidly extended and energetically drawn back against the body which results in the rotation of the body. After some time these motions become slower and finally cease. The organism is more readily found in fresh preparations, especially if a small amount of eosin is added as suggested by Drescher and Hopfengärtner.[9] For detailed morphological studies, stained preparations are necessary for which purpose either iron-hematoxylin or Giemsa following air-drying of thin smears and fixation in methyl alcohol can be used.

Although the greater part of the credit for first recognizing trichomonads as the causative agent of early abortion with the character of an infectious disease belongs to Riedmüller, yet much credit is also due Abelein,[10] who worked diligently and incessantly to show that many of the abortions occurring in southern Germany were not due to Bang's disease or to vaginal catarrh but to trichomonads. In such cases he found pyometra and early expulsion of the fetus. He also found trichomonads in the preputial sac of the bulls where they were most abundant in cases of preputial catarrh. He deposited trichomonads upon the *os uteri externum* of healthy cows at the time of service and produced early abortion followed by sterility. The publication of these findings immediately set a large number of workers checking these findings with the result that today we have a fairly clear picture of the disease.

Soon after Abelein published his first findings, Emmerson[11] found flagellates with an undulating membrane in the whitish-colored, mucus-like vaginal discharge of a naturally infected cow in Pennsylvania, but did not observe abortion in this cow. Wenrich[6] identified this protozoan as belonging to the genus *Trichomonas* and later described it with Emmerson as *Tritrichomonas foetus* (Riedmüller). Emmerson reports that he successfully transmitted his trichomonads to another healthy cow in which they could be demonstrated from the tenth to the 35th day. The trichomonads remained alive in the discharges kept at room temperature for eleven and 19 days, respectively.

Following the finding of trichomonads in America by Emmerson, other American investigators began to look for them, with the result that the disease has been observed by McNutt, Walsh

and Murray,[12] by Cameron, Fincher and Gilman[13] and by Cameron.[14] In Europe the disease was soon recognized as being widespread in southern Germany, northern Italy, Switzerland and Austria. The widespread appearance of the disease in those countries could not fail to attract the attention of many research workers, with the result that our knowledge of the disease has been materially advanced within the last three years.

It was early recognized that pure cultures were highly desirable and needed if progress in developing proof that *Trichomonas foetus* was responsible for the pyometra and early abortions. Riedmüller[17] was the first to attempt to obtain trichomonads free from bacterial contamination. He experimented with small laboratory animals and found that the intraperitoneal injection of trichomonads was fatal to guinea pigs and that the trichomonads could be found in pure culture in the peritoneal exudate. Abelein[9] found that intra-uterine injection of pregnant guinea pigs with uterine secretion containing trichomonads frequently yielded pure cultures of the organisms. Witte[7] succeeded in obtaining pure cultures *in vitro* from aborted bovine fetus and bovine uteri which he cultured on blood bouillon and serum bouillon. With these cultures he later[16] succeeded in infecting vaginally and producing abortion in guinea pigs and rabbits. Guinea pigs also aborted when injected intraperitoneally. Following abortion, they usually died showing a purulent peritonitis. Trichomonads were found in the peritoneal exudate and the large parenchymatous organs. Witte declares the cultivation of trichomonads on blood bouillon is quite successful and frequently gives positive findings when direct microscopical examination is negative.

Witte[15] later extended his artificial vaginal infection experiments with his pure artificial cultures *in vitro* to three heifers and produced an acute vaginitis and sterility. After healing of the acute vaginal catarrh, a swelling of the follicles was observed. Since another cause for the sterility of the three heifers could not be ascertained, he ascribed it to the pathogenicity of the trichomonads. This view is supported by the fact that the organism is found in aborted fetuses often in pure culture. He found trichomonads in twelve cases in aborted fetuses in the absence of pathogenic bacteria and found them twice in pure culture.

Gehring and Murray[8] used Witte's method among others for culturing trichomonads. They, however, had better results by growing the trichomonads on Locke's egg-blood media to which they added 1:1,000 aqueous crystal violet to restrict bacterial growth. They did not succeed in getting cultures free from bacteria.

Trichomonads may be found in pure culture in aborted fetuses. Such pure cultures were reported by Drescher,[19] one case; Pfenniger,[3] two cases; Abelein,[10] one case, Riedmüller,[4, 17] four cases, and Witte,[15] two cases. Riedmüller[17] tried the complement-fixation test on two cows that had recently suffered a trichomonas abortion in the fetuses of which trichomonads appeared in pure culture. He prepared his antigen by injecting guinea pigs intraperitoneally and upon their death collected the trichomonads from the peritoneal fluid. He obtained positive complement-fixation test in both cows.

Symptoms

Our knowledge concerning trichomonas abortion in cattle has been materially enriched in the last two years, especially by European workers, and much has been written on it. More or less detail on the different phases of the disease will be found in the writings of Abelein,[10, 20] Küst,[18, 19, 21, 27, 28] Dreschner and Hopfengärtner,[9] McNutt, Walsh and Murray,[12] Cameron, Fincher and Gilman,[14] Schumann,[22] Cameron,[14] Kubesch,[23] Brauer,[26] Heymach,[25] Schaaf,[24] and Daust.[29]

To review critically all these writings would lead us too far afield at this time. Suffice it, therefore, that a general picture of their findings be given.

Pyometra and early abortion must be considered as typical for trichomonas abortion in cattle. Practically all writers on the disease agree that it is readily transmitted from cow to cow by the diseased bull and from herd to herd by a diseased bull or cow. No other mode of transmission has thus far been found, although the possibility of one is suggested in the finding of Daust, as quoted by Küst,[19] that the trichomonads can live in urine for ten days. In the bull, upon first being infected by service of an infected cow, there develops, one or two days later, an inflammation of the prepuce which may cause swelling and intense reddening. There is then a more or less profuse, muco-purulent discharge from the prepuce. During the acute stage, the bull is often slow to serve, but otherwise shows no systemic symptoms. Microscopical examination of the discharge from the prepuce shows many actively motile trichomonads. Closer examination of the highly reddened preputial mucosa in the acute stage will show the development of numerous nodules about the size of a millet-seed.

The acute inflammation soon begins to improve, the preputial mucosa gradually tends to assume its normal color again, but the nodules will persist for a long time. At this stage, externally manifested symptoms are no longer detectable and the bull may

pass as sexually healthy and trichomonads are now very hard to find in the secretions or even are absent. Such bulls are still capable of transmitting the infection and will continue to do so even after months of sexual rest. Bulls once infected are always dangerous to use, for even though the trichomonads may no longer be present on the preputial mucosa yet other parts of the genital tract may harbor them and such bulls are chronically affected.

Thus, Feiling (quoted by Küst[28]) could repeatedly demonstrate trichomonads in the ejaculate of chronically affected but clinically recovered bulls. Trichomonads could not be found, however, at every examination. Furthermore, a number of cases of pure trichomonad infection could be produced in cows artificially inseminated with the semen of such chronically affected, clinically recovered bulls. These facts may help to explain why not all cows served by chronically affected bulls become diseased. Cameron, Fincher and Gilman[13] report finding organisms resembling trichomonads in the head of the epididymis of a bull infected with trichomonads but could not find such organisms in the tail of the epididymis, testes or urethra.

In the cow the symptoms shown depend much upon the stage of infection in the bull at the time of service. Should the bull be in the acute stage of infection, with many trichomonads in the preputial secretion, then the cow also is very likely to develop, in the course of a day or two, an acute form of the disease, manifesting itself in a muco-purulent discharge from the vulva as the result of an acute vaginitis, swelling of the vulva, and uterine catarrh. The vaginal mucosa becomes highly reddened and there develop numerous nodules on it similar to those found in the bull. The vaginitis soon disappears, but the nodules will remain for a long time. These conditions usually prevent conception, so that such cows come in heat regularly and, especially during heat periods, show a muco-purulent discharge from the vulva which originates in the uterus. Examination of such discharge reveals numerous trichomonads.

In the interval between heat periods, an examination of the vaginal mucosa frequently does not show the parasites. If the bull is in the chronic stage of infection, an acute vaginitis may also develop but more frequently it does not and in such case no clinical symptoms of disease develop. The cow either did not become infected at all, conceived and will calve normally, which is rare, or she may become infected and may show a few trichomonads in the vaginal secretions at intervals during the entire period of pregnancy.

In another group conception has taken place in spite of the simultaneous infection. The developing fetus dies as a result of the advancing and spreading infection throughout the uterus, but is not aborted and undergoes maceration accompanied by pyometra. The corpus luteum persists, the cervical canal remains closed and vaginal examination reveals normal conditions. Due to the absence of recognizable external symptoms and the advancing accumulation of pus in the uterus, the cow is considered pregnant by the owner. Rectal examination shows both horns of the uterus equally large, fluctuating and signs of pregnancy absent. This pyometral condition may be interrupted at any time, large quantities of the thin, yellowish-gray, egg-soup-like or pea-soup-like pus evacuated, in which many trichomonads are present. In some cases small coagulated lumps are found in the pyometra pus which is of a peculiar sweetish odor and never putrid. In some cases an almost clear, watery exudate is encountered.[23] If natural evacuation does not occur, the condition continues until discovered by a chance examination or such examination is requested by the owner because the condition has persisted greatly beyond term. Abelein[10] could interrupt this condition in 95 per cent of the cases by expression of the persistent corpus luteum.

In still another group conception has occurred in spite of infection. They show only a temporary vaginitis and, because of conception, estrum does not appear. After six to 16 weeks, sometimes even later, a marked muco-purulent discharge may suddenly appear which is usually soon followed by abortion. In the expelled fetuses, which usually have a rather fresh-looking appearance, and in the muco-purulent discharge, in the frequently markedly pale and anemic, and often peculiarly knotted fetal membranes and in the amnionic fluid, numerous actively motile trichomonads may be found. Estrum often occurs two to three days after abortion and, especially if the abortion was not observed, such animals are not infrequently bred at this period. Such cows excrete the trichomonads for a number of days following abortion and naturally constitute a dangerous source of infection for the bull.

If the abortion was complete, that is, if the fetus, placenta and amnionic fluid were completely expelled, complete recovery will take place in the course of several weeks. Trichomonads could be found in the discharges by Küst and his co-workers up to the ninth day following such complete abortion. The estrual cycle will later become regular again and, at the end of two estrual cycles, the cows will usually conceive at the first service.

If the abortion was not a complete one, if all or part of the placenta and amnionic fluid is retained, purulent endometritis follows and in all such cases prolonged intensive treatment is necessary. Later estrum is regular again but during estrum a more or less marked muco-purulent discharge is noted.

Upon clinical examination one finds a more or less marked vestibulitis and vaginitis with a marked thickening of the mucosa and nodule formation, especially around the external os. One finds here what Abelein[10] calls a rasp-like or grater-like condition of the mucosa which may be considered as specific for trichomonas infection. Usually a diseased condition of the ovaries exists simultaneously. In all cases the diagnosis of trichomoniasis of cattle must be based upon a systematic examination and the demonstration of the causative organism.

Treatment

Naturally many minor variations from the above typical cases and grouping have been observed, but to record all is beyond the scope of this paper. The treatments used are probably as numerous as individual workers. In the cow, the German veterinarians, in addition to the usual sanitary measures, uniformly recommend the introduction of about 100 cc of Lugol's solution into the uterus after it has been flushed with some other fluid, usually saline solution, or the use of chinosol 1:1,000 or entozon. As a rule only the cases developing an endometritis call for prolonged and persistent treatment. The other cases usually recover spontaneously within a few weeks and will then conceive again and calve normally unless again infected at service.

In the case of bulls the consensus is that as a rule they are incurable. For apparently recovered bulls the only safe procedure recommended is to subject them to a number of test breeding trials on virgin heifers. It is possible that artificial insemination in the case of valuable bulls not showing infected semen but carrying infection of the prepuce, when conducted under proper precautions, may prove of value during the critical breeding season or until such time as a replacement sire may be obtainable. Since not all cows become regularly infected at every service, the proper procedure to adopt must be determined by local conditions of operation.

Our Own Observations

This disease has been found in a large dairy herd in Texas which is now under the observation of the writer. This herd, consisting of Jerseys and Holsteins, had been freed of Bang's

disease a number of years ago and has been kept free. Late last summer, a series of six abortions occurred in this herd, which were not reported at the time, as a result of which an examination of the fetuses was not possible. The agglutination test for Bang's disease remained negative in these cases. Instructions were therefore given to bring the next aborted fetus to the laboratory for examination. Trichomonads were found in this fetus. Later, two more abortions occurred and trichomonads were found in both cases. Breeding operations were immediately stopped.

An inquiry as to the clinical symptoms shown by the bulls during the last year revealed that recently one, a young Holstein bull, had shown a marked discharge from and swelling of the prepuce. Since trichomonads were found in both the Jersey and Holstein females, it was evident that trichomonas infection could be expected in the bulls of both breeds. Examination of the bulls under epidural anesthesia was therefore undertaken with unsheathing of the penis. These examinations showed all seven bulls to have more or less inflammation of the preputial mucosa and many nodules the size of a millet-seed. The severest inflammation and the greatest number of nodules were found in the Holstein bull that recently showed a preputial discharge and swelling of the sheath. The nodules were more or less highly reddened and many exhibited a gray center.

Under the application of a 1½ per cent oxyquinoline sulfate solution, with light rubbing of the mucosa with the fingers, many nodules would assume a hemorrhagic center or would even bleed. Four treatments, about a week to ten days apart, have thus far been given. At the time of the third treatment, improvement in some of the bulls was manifest. At this time numbers of tiny gray specks were found on the last two bulls treated for the day. At the fourth treatment it became obvious that these tiny gray specks, hardly larger than a pin-point and very sharply defined, could easily have been overlooked in the other bulls at the third treatment, because we could not see them in spite of close inspection before all the mucus was washed from the prepuce. Only after this was done and especially when the sun was shining directly upon the surface being inspected did they stand out prominently. In the shade they could hardly be seen. Thus, at the fourth treatment, we found a decrease in the larger nodules. It is the writer's opinion that the latter are small lymph follicles and that they probably are the location at which nodules may develop.

Before the first treatment was begun on the bulls, the penis and prepuce were thoroughly washed with sterile Locke's solu-

tion under rubbing with the fingers in order to remove and collect in it any trichomonads that might be present. The fluid was then gently centrifuged and the sediment examined for trichomonads. None were found. The fluid was then allowed to stand for 24 hours and then examined again. Again the results were negative.

In the females in which trichomonads were found the larger nodules also were present. An opportunity to examine them for the tiny gray specks has not yet been afforded.

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Discussion

Dr. B. A. Beach: I agree with Dr. Schmidt that the disease is undoubtedly more widespread than most of us realize. I know we are encountering considerable of it in Wisconsin, and on the few bulls which we have been privileged to investigate, a period of rest apparently effected recovery. In two of the three, we were positive of it because they are in service now in negative herds. There is evidently considerable immunity developed; how long an association with the organisms is necessary, we do not know. All our observations are field observations. A bull was purchased from another state—it happened to be Dr. Murray's state, and I am glad to tell him about it now—and introduced into a herd in southern Wisconsin. While a positive diagnosis of trichomonad infection up to that time had not been secured from that herd, it has been now for some considerable time. However, up to that time it had not and they had considerable breeding trouble, as they have had in a number of the larger herds. At the time this bull was purchased, a number of virgin heifers also were purchased, and the older sire was used on the main herd and on about half of the virgin heifers. The other half of the virgin heifers were served by a young bull. I think I am right when I state that nearly all of the virgin heifers bred to the old bull aborted. They came in heat at irregular intervals, and although all of them were not known to have aborted, they probably did. A fetus examination disclosed large numbers of the organisms. There was no trouble in the old cows which were served at the same time, and the other half of the virgin heifers calved normally. That indicated considerable resistance to the infection on the part of the old cows. Yet the same sire caused a lot of trouble in the heifers.

Dr. G. Dikmans: Dr. Schmidt, I would like to ask whether or not these nodules, as described, are found in the acute cases of the disease, or only in the chronic cases?

We had two cases relating to this. Dr. F. W. Miller, of the Bureau of Dairying, had discovered them in the herds kept by his department for experimentation. We went over those cases very carefully, we opened up the vaginas, and we were unable by sight or touch to find any of these nodules as described. I refer to the descriptions given by Abelein and Fincher in their latest articles. I would like to know whether these nodules are a development of the acute stage, or whether they appear later, in the chronic stages. Perhaps I should not say "chronic," but I mean after the disease has been developing for some time.

Dr. Schmidt: Abelein describes them as characteristic of early abortion. He describes them as following especially those cases in which there is a long period of discharge of the material from the vagina, giving the trichomonads a good opportunity to become established in the mucosa.

In the bulls, we found them right away, so there is no mistaking these nodules, once it is indicated. They show a gray center in the beginning. In those instances where we have observed these tiny gray specks, I think it is nothing but the center that you see in the nodules before they begin to develop.

Dr. M. G. Fincher: I should like to say something with reference to just how pathognomonic the roughing of the mucosa of the vagina

is. I think in most instances you will find the roughing there, which means you have the trichomonad disease, providing the proper test is made. The absence of those nodules, however, does not mean that the cow does not have trichomonad disease. I have not found them in the bull. Two autopsies of the bull failed to show any nodules. Perhaps they had reached a stage where they would not show.

DR. SCHMIDT: We found them in all seven of the bulls, but they are gradually disappearing now. We began treatment on June 28. We have given them four treatments, and within 30 days we could notice a decrease in the number of nodules.

DR. J. B. HAGENBUCH: Did I understand you to say that you had several bulls that had it and then cleared up and were being used in breeding?

DR. BEACH: Apparently that has happened. I know one of them is in service today. I think one of them is in a negative herd, one in which no trouble has ever been experienced. He has been in there for a couple of years or more. Whether or not they all recover, I do not know. We have not had an opportunity to observe enough of them.

DR. HAGENBUCH: In our experience, they do not recover. We have not felt safe enough to breed them, except when they have been douched prior to breeding, and they are a great risk to us then.

DR. BEACH: Were your bulls retired from service for six months to a year?

DR. HAGENBUCH: We experimented with one bull for a year. Of course, the man douched that bull prior to breeding, but that bull still was a risk at the end of a year.

DR. BEACH: He had not been rested completely for six months to a year?

DR. HAGENBUCH: No. He received about two months rest.

DR. BEACH: I hesitate to recommend that, but apparently there seems to be something in it. If there is complete rest for six months or more, at least some of them will recover, or apparently recover. I do not know what the percentage is. We have not had the opportunity to study enough of them.

DR. HAGENBUCH: That has been your experience?

DR. BEACH: Yes. I might say something about the history of one of these bulls. He was retired from service for six months, or longer, and then was put into a sale and sold, which should not have occurred. That bull is in service today. He has been used on a number of different farms and trichomoniasis was definitely diagnosed in two herds in which he was used before he was put into them.

DR. FINCHER: How carefully were these herds looked into to see if he had not taken it to them?

DR. BEACH: Well, as I say, I do not know the percentage of bulls which recover, but apparently at least two of them have, and this one bull has been used in at least two herds. We are absolutely unable to find any history or anything to indicate that the disease is present in either one of these herds.

Dogs Destroyed in Flooded Areas

According to a note in *Dog World*, it is estimated that more than 10,000 dogs were destroyed by army officials in the flooded areas along the Ohio River during January and February.

He who is too powerful is still aiming at that degree of power which is unattainable.—Seneca.

THE PRESENCE OF AVIAN TUBERCLE BACILLI IN THE LYMPH-NODES OF CATTLE*

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Although Wisconsin has been an accredited state for bovine tuberculosis since 1932, yet 0.24 per cent of the cattle tested in 1935 reacted to tuberculin. About 50 per cent of these animals showed tuberculous lesions upon autopsy, while the other 50 per cent showed no visible lesions of tuberculosis or skin lesions only. Hastings, Wisnicky, Beach and McCarter[1] found, in examining the lymph-nodes of an experimental group of these no-visible-lesion, tuberculin-reacting cattle, that the bovine tubercle bacillus was absent but there were present certain of the so-called saprophytic species of *Corynebacterium* and *Mycobacterium* and also avian tubercle bacilli in one specimen. Undoubtedly some bovine tuberculosis still exists in Wisconsin, but it seemed possible that some part of the tuberculin reactions in lesion as well as in no-visible-lesion cattle might be explained by the invasion of the tissues by organisms other than the bovine tubercle bacillus. Accordingly, we decided to examine the tissues of tuberculin-reacting lesion cattle.

EXPERIMENTAL

Lymph-nodes from tuberculin-reacting cattle, showing only slight lesions localized in the lymph-nodes, were obtained from Wisconsin packing plants. This material was kept in a frozen condition until the records on the postmortem inspection and on the herd record of tuberculin testing were obtained. The nodes of cattle from herds which had been in existence for a period of several years, with a history of only a few reactors, were desired for examination, since there was less probability that lesions from such animals were caused by bovine tubercle bacilli. The records showed that only a few of the lesions were coming from animals in herds with a small number of reactors. Therefore, some of the lesions from cattle in herds showing a larger number of reactors were used to check our methods for the detection of the bovine tubercle bacilli.

A small part of the tissue containing visible lesions was ground with sand in a mortar, suspended in saline solution, filtered through gauze, and inoculated into guinea pigs; another part of

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the tissue was cut in small pieces, treated with antiformin, and seeded on culture media. Two guinea pigs and approximately 40 tubes of media were used for the tests on lymph-nodes from one animal.

During the first part of the experiment, Dorset's coagulated egg medium* and Corper and Uyei's potato medium† were used. The results are given in table I. It will be seen from these data that no bovine tubercle bacilli were cultured from any of 23 samples examined, even though the results of guinea pig inoculation indicated the presence of bovine tubercle bacilli in 15 of these samples. Subsequently, Herrold's egg-yolk agar‡ was used in addition to Dorset's medium, instead of the potato medium. The use of the coagulated egg medium was continued since the growth of acid-fasts on it would be presumptive evidence that the organisms were other than bovine tubercle bacilli. In table II are the data on the examination of a second group of samples.

Acid-fast organisms obtained upon culture were identified by cultural characteristics and by the results of the guinea pig inoculation. The identification of the avian tubercle bacilli was verified by the inoculation of rabbits and guinea pigs with suspensions of the cultures. In every case in which cultures having the characteristics of mammalian tubercle bacilli were isolated, the guinea pigs developed tuberculosis. The livers, spleens and lungs of one of each pair of tuberculous guinea pigs were cultured, but the results were all negative during the first part of the experiment when the coagulated egg medium was used. When the uncoagulated egg-yolk medium was used during the latter part of the experiment, results were obtained in 100 per cent of the attempts and bovine tubercle bacilli were isolated from the tuberculous tissues of ten guinea pigs.

*Dorset's egg medium:

24 eggs
300 cc distilled water
60 cc glycerin

Sterilized in autoclave.

†Corper and Uyei's potato medium:

Potato cylinders washed in tap water, soaked in 1 per cent sodium carbonate solution containing 0.001, 5 per cent crystal violet for 2 hours, wiped dry, placed in test-tubes containing glass rods to support them and 5 per cent glycerin nutrient broth added to immerse the butts. Sterilized by autoclave.

‡Herrold's egg-yolk agar:

3 gm Liebig's beef extract
10 gm Peptone (Difco)
5 gm NaCl
10 gm agar
1,000 cc distilled water

pH adjusted to 7.5. Subsequent to sterilization, one egg yolk added aseptically to 150 cc agar.

The data in tables I and II show two things: first, that Herrold's egg-yolk agar is suitable for the cultivation of bovine tubercle bacilli, and second, that avian tubercle bacilli may be present in tuberculous lymph-nodes of tuberculin-reacting cattle. Since these two findings are distinct, they will be discussed separately.

Discussion

Culture media for the isolation of bovine tubercle bacilli: The coagulated egg medium and the potato medium proved to be totally unsatisfactory for the cultivation of bovine tubercle bacilli from tuberculous bovine tissues and from tuberculous guinea pig tissues. Herrold's uncooked egg-yolk agar medium was satisfactory in the majority of cases (six out of eight) for isolation from the bovine lesions, and in all cases (ten) for the isolation of bovine tubercle bacilli from the tuberculous tissues of the guinea pigs. Feldman[2] obtained results comparable to ours in using Herrold's medium for isolating bovine tubercle bacilli from the tissues of cattle, but failed in three of 17 attempts to isolate bovine tubercle bacilli from the tissues of guinea pigs. Feldman used a method differing from ours for the treatment of the guinea pig tissue before culturing (we used the same treatment as for the bovine lymph-nodes), and he also modified the water content of the medium.

The presence of glycerin was partially responsible for the unsuitability of the coagulated egg medium and the potato medium. Corper[3] showed that glycerin is bacteriostatic to small inocula of bovine tubercle bacilli. We have found it possible to carry our stock cultures of bovine tubercle bacilli on the coagulated egg medium with glycerin, and also previously in several instances we have isolated bovine tubercle bacilli from the tissues of tuberculous guinea pigs on this medium. But in these cases the bacteriostatic effects of the glycerin were probably overcome because relatively large inocula were used. (Microscopic examination reveals many more acid-fast organisms in the spleens of guinea pigs inoculated with bovine tubercle bacilli than in the tuberculous bovine tissue.)

The significance of the presence of avian tubercle bacilli in the lymph-nodes of cattle: Several workers have reported the isolation of avian tubercle bacilli from the tissues of cattle. The literature on the infectivity of the avian tubercle bacillus for cattle is adequately reviewed by Feldman.[4] Our results are reported not only because they give some information concerning the incidence of infection of Wisconsin cattle with avian tubercle

TABLE I—*Examination of autopsy material from tuberculin-reacting cattle cultures made on coagulated egg medium and potato medium containing glycerine.*

Herd History and Autopsy Results					Results of Laboratory Tests on Lymph-Nodes	
Herd	Animals Tested	Animals Reacted	Animal	Lesions*	Culture†	Guinea Pig Inoculation‡
1	27	7	1	sl, med, cc	—	+
			2	wm, med, cc	—	+
2	38	5	1	sl, cerv-mes, cc	—	+
3	18	3	1	wm, bron, c, sl, med, c	—	+
4	19	6	1	wm, cerv-bron-med, cc	—	−
5	23	5	1	sl, lungs, cc	—	+
6	26	4	1	sl, mes, cc	Avian t. b.	−
7	25	7	1	sl, bron-med, cc	—	+
			2	sl, bron, cc	—	+
8	62	2	1	sl, med, cc	—	+
			2	sl, bron, cc, ext, mes, cc	—	+
9	22	4	1	sl, med, cc	—	+
10	37	2	1	sl, mes, cc	—	+
11	14	1	1	sl, bron-med, cc	—	+
12	31	31	1	ext, cerv, cc, sl, med, cc	—	+
			2	sl, bron, cc	—	+
			3	sl, bron, cc	—	0
13	19	19	1	sl, bron-med, cc	—	0
			2	sl, bron-med, cc	—	0
			3	sl, med, cc	—	0
14	49	1	1	sl, mes, cc	—	−
15	23	1	1	sl, bron-med-mes, cc	Avian t. b.	−
16	19	1	1	sl, med, cc	—	+

*sl = slight; wm = well-marked; ext = extensive; cerv = cervical; bron = bronchial; med = mediastinal; mes = mesenteric; c = caseous; cc = caseo-calcareous.
†— = negative for the growth of acid-fast bacteria.
‡+ = tuberculosis found upon autopsy; — = no tuberculosis found upon autopsy; O = test not made.

TABLE II—*Examination of autopsy material from tuberculin-reacting cattle cultures made on uncoagulated and coagulated egg media.*

HERD HISTORY AND AUTOPSY RESULTS					RESULTS OF LABORATORY TESTS ON LYMPH-NODES	
HERD	ANIMALS TESTED	ANIMALS REACTED	ANIMAL	LESIONS	CULTURE	GUINEA PIG INOCULATION
17	20	1	1	sl, bron, cc*	—	+
18	47	1	1	sl, bron, cc	mammalian t. b.	+
19	11	1	1	sl, med, cc	avian t. b.	—
20	22	2	1	sl, med, cc	—	+
21	22	22	1	sl, bron-med, cc	mammalian t. b.	+
			2	sl, med, cc	—	0
			3	sl, med, cc	mammalian t. b.	+
22	22	5	1	sl, bron, cc, wm, mes, cc	mammalian t. b.	+
23	30	1	1	sl, bron, cc	mammalian t. b.	+
24	20	4	1	wm, med, cc, sl, lungs, cc	mammalian t. b.	0
			2	ext, cerv, cc, wm, bron-med, cc	mammalian t. b.	+

*For key to abbreviations, see table I.

bacilli, but also because they indicate the relation which infection with avian tubercle bacilli bears to tuberculin reactions in cattle.

As shown in tables I and II, three out of 34 cattle, whose lymph-nodes we examined, were infected with avian tubercle bacilli. The lesions in these three cases had the same gross pathology as those in the tissues from which bovine tubercle bacilli were cultivated. The casual relationship of the avian tubercle bacillus to the lesions in the nodes from which they were isolated has not been proved. In none of the three cases, however, did the guinea pigs injected with the tissue emulsions develop tuberculosis, indicating that a synchronous infection with bovine and avian tubercle bacilli did not exist.

Since avian tubercle bacilli do not produce a generalized and progressive tuberculosis in cattle, their occurrence in cattle is significant mainly from the standpoint of their interference with the interpretation of a reaction to tuberculin in diagnosing tuberculosis caused by bovine tubercle bacilli. These three cattle which were infected with avian tubercle bacilli were slaughtered because they reacted to mammalian tuberculin during the routine testing. No information is available on the details of the extent of the reactions in these cases, but it must be assumed that the reactions were typical. Schalk and co-workers[5] obtained in cattle experimentally exposed to infection with avian tubercle bacilli only one positive reaction to mammalian tuberculin and three questionable reactions. They concluded:

> The failure, therefore, to find visible lesions of tuberculosis upon autopsy of cattle, which have been condemned because of positive tuberculin tests in routine eradication work, cannot be attributed to a previous exposure of those animals to tuberculous chickens.

Feldman[4] reported that of three cattle from whose tissues he cultivated the avian tubercle bacillus, one had given a typical reaction to mammalian tuberculin and two had given atypical reactions.

In man, reactions to the avian and mammalian tuberculins seem to vary quantitatively rather than qualitatively, as shown by a study made by McCarter, Getz, and Stiehm,[6] in which tuberculins chemically standardized according to the method of Seibert[7] were used. That is, an individual may react to both mammalian and avian tuberculin, but more markedly to one than to the other. It would seem logical, therefore, to expect that in cattle also reactions to avian and to mammalian tuberculins could be quantitatively rather than qualitatively specific;

but it would be impossible to prove this by the ordinary tests with avian and mammalian Old Tuberculins which are not standardized and vary widely in content of active principle.

No satisfactory method for differentiating cattle reacting to tuberculin because of infection with avian tubercle bacilli from those reacting because of infection with bovine tubercle bacilli is apparent at the present time. For reasons explained above, the testing of cattle with avian tuberculin as well as with mammalian tuberculin would be useless, since it is possible that an animal infected with bovine tubercle bacilli might react more to avian than to mammalian tuberculin. Again, it is not possible to apply here the method used to reduce the number of no-visible-lesion, tuberculin-reacting cattle; namely, animals giving tuberculin reactions deviating in some degree from the normal, in herds which have no history of tuberculosis and in which there are only one or two reactors, are not removed for slaughter. (Animals giving typical reactions are always removed.) It is very possible that there would be a history of tuberculosis in those herds from which animals infected with avian tubercle bacilli came, since in the lesions we examined those apparently caused by avian tubercle bacilli were indistinguishable from those caused by bovine tubercle bacilli. Although our data are too few to indicate the probabilities, in one case the animal infected with avian tubercle bacilli came from a herd with four reactors of 26 tested, and in the other two herds the animals infected with avian tubercle bacilli were the sole reactors. The possibility exists, therefore, that several reactors infected with avian tubercle bacilli may occur in a herd.

The practical import of this work is that the tuberculosis of hogs and chickens caused by the avian tubercle bacillus must be controlled before tuberculosis of cattle can be eradicated. The prevalence of tuberculosis in Wisconsin flocks and the effect this is having in increasing the tuberculosis in Wisconsin hogs has been stressed by McCarter, Beach and Hastings.[8] Sensitization by the avian tubercle bacillus is undoubtedly responsible for a part of the residuum of tuberculin-reacting cattle, both lesion and no-visible-lesion animals, in Wisconsin. The ratio of the number of cows reacting because of infection with avian tubercle bacilli to the total number of reactors has been increasing as the number of animals infected with bovine tubercle bacilli has been reduced, and will probably continue to increase until measures are taken to eradicate tuberculosis of chickens and hogs.

CONCLUSIONS

1. Of the tuberculous lymph-nodes of 28 tuberculin-reacting cattle examined by cultural methods and by guinea pig inoculation for the presence of acid-fast microörganisms, three were found to be infected with avian tubercle bacilli, 23 with mammalian tubercle bacilli, and negative results were obtained on the remaining two.

2. It is probable, therefore, that infection with avian tubercle bacilli is responsible for a part of the tuberculin reactions in lesion as well as in no-visible-lesion cattle in Wisconsin.

3. Herrold's egg-yolk agar medium has been found to be completely efficient for the culture of bovine tubercle bacilli from tuberculous guinea pig tissues, but is not quite so satisfactory as the inoculation of guinea pigs for the detection of bovine tubercle bacilli in the tuberculous tissues of cattle. The factors responsible for the superiority of Herrold's egg-yolk agar over the other media used have not been studied.

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State Board Appointments

Governor Horner has appointed Drs. W. H. Shaw (Chi. '10), of Pawnee, and William W. Warnock (Ont. '04), of Aledo, to membership on the Illinois State Board of Veterinary Examiners. The other member of the Board is Dr. L. A. Merillat (Ont. '88), of Chicago, who has been serving alone for several years pending appointments to fill the two vacancies.

Dr. Nevin S. Craver (O. S. U. '21), of Youngstown, Ohio, has been appointed a member of the Ohio State Board of Veterinary Examiners by Governor Davey, to succeed Dr. C. McCandless (Chi. '15), of Salem, whose term had expired.

THE PATHOLOGY OF CROTALARIA SPECTABILIS ROTH SEED POISONING IN THE DOMESTIC FOWL*

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Crotalaria spectabilis seed poisoning in the domestic fowl has been reported by Thomas.[1] Since the completion of his investigation, many cases of *C. spectabilis* seed poisoning have been presented for observation and postmortem examination. Our attention was directed to one bird in which a severe anemia was present.

As Thomas failed to observe anemia in the birds used in his experiments, it was deemed advisable to conduct further studies on the pathology of this type of poisoning in the domestic fowl, particular attention being directed to the blood picture and histopathology.

EXPERIMENTAL DATA

Nineteen birds were force-fed *C. spectabilis* seed, as indicated in table I. Total and differential blood-cell counts as well as the hemoglobin readings (Sahli) are given.

Symptoms: Birds affected with the acute type of *C. spectabilis* poisoning usually have congested comb, full crop, ruffled feathers, listless and droopy appearance and a greenish, yellow diarrhea. In the chronic type of poisoning the comb becomes more or less pale, depending on the severity of the anemia present; the crop is often full; the diarrhea is usually copious; more or less emaciation occurs; and in the later stages the bird becomes droopy and the feathers often are ruffled.

Gross lesions: The outstanding gross lesions of the acute type of *C. spectabilis* poisoning are innumerable petechiae in the serous membranes of the abdominal and thoracic cavities, as well as in the visceral fat and a liver normal in size but characteristically dark and marbled in color.

In the acute type of poisoning, the crop usually is full and contains seeds of *C. spectabilis.* Congestion of this organ often is noted. The gizzard may also contain seeds of *C. spectabilis.* The interior of this organ often is discolored by bile. The bulbous portion of the gizzard occasionally shows necrosis and a separation of the lining from the thin muscular wall. The intestines may show slight catarrhal inflammation and usually are congested. No consistent lesions seem to develop in the proventriculus. Occa-

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sionally a few petechiae appear at the union of the proventriculus and gizzard. Petechiae invariably are present in the myocardium and coronary fat (fig. 1). The serous membranes of the abdominal and thoracic cavities and the visceral fat contain innumerable petechiae. In some of the acute cases serous fluid, 100 to 200 cc, is found in the abdominal cavity. A slight serous pericarditis also may occur. The liver appears dark and marbled in color. There are, however, a few pale areas present. Slight enlargement and congestion of the kidneys occurs.

FIG. 1. Characteristic cardiac lesions of acute *Crotalaria spectabilis* poisoning in the chicken.

In the chronic type of *C. spectabilis* poisoning the spleen usually shows atrophy and is pale in color; congested areas may occur. Often the crop is full and the mucous membrane is white in color. Necrotic enteritis invariably occurs in the duodenum, the serosa of the affected portion being thickened and gray. Caudally the remainder of the small intestine is usually atrophied. The myocardium is soft and flabby. Serous pericarditis with occasional adhesions often occurs. Petechiae which are so characteristically present in the myocardium, the serous membranes of the abdominal and thoracic cavities and the visceral fat in acute

cases are very few in number and in the more chronic cases are entirely absent. The development of an extensive ascites often occurs in chronic cases. This condition seems to be associated with the development of anemia. The liver may appear as in the acute type of poisoning. In cases in which ascites is present this organ often is covered with a coagulum. In many cases the surface of the liver is uneven and shows a few pale areas. Scattered gray foci commonly are found on the surface. The gall-bladder usually is enlarged. The kidneys appear pale and in many cases atrophied. The subcutaneous fat often is darker than normal.

Histopathology: The capsule of the spleen is thickened irregularly. The smooth muscle fibers in the capsule as well as those of the capsular arteries are in a state of cloudy swelling. The capsule often is edematous and occasionally small hemorrhages are present. The spleen invariably shows congestion, tending to be less marked in the more chronic cases. A few small foci of necrosis often are observed. The splenic cells in several instances show cloudy swelling and occasionally the organ shows a reduction in splenic cells, particularly near the capsule, to the extent that only the reticulum of the organ remains. Small foci of proliferation of connective tissue and perivascular foci of proliferation of histiocytes are not uncommon.

The surface epithelium of the crop, as well as the smooth muscle of the walls, is in a state of cloudy swelling. A few small intermuscular hemorrhages occur.

Cloudy swelling occurs in the surface and glandular epithelium as well as in the epithelium of the collecting ducts of the proventriculus. Cloudy swelling and edema are prevalent in the muscular walls of this organ. Foci of histiocytes, rather large but often loosely arranged, occur frequently in the submucosa and interglandular tissue. A few small hemorrhages also occur occasionally in the submucosa.

The serosa of the duodenum usually is thickened and edematous. Occasionally small subserous hemorrhages occur. The musculature of the intestinal wall is thickened irregularly, the muscle cells showing cloudy swelling. In the zone of greatest thickness the muscle cells often are almost entirely devoid of cytoplasm. Slight edema often occurs between the muscle fibers. Edema also is common in the submucosa, the muscularis mucosa usually showing considerable cloudy swelling. Necrosis of the extremities of the villi occurs. The remainder of the epithelium of the villi, as well as the glandular epithelium, is in a state of cloudy swell-

TABLE I—*Total and differential blood counts of birds experimentally affected with Crotalaria spectabilis Roth seed poisoning.*

Bird	Seeds Fed	Date Fed	Date Blood Count	Date Autopsy	Erythrocytes	Leukocytes	Hemoglobin (Sahli)	Polymorphonuclears	Lymphocytes				Basophiles	Monocytes	Myelocytes	Polychrome Erythrocytes	Basophile Erythrocytes
									Mature	Vacuolar	Immature	Degenerative					
418	10 every	10–30	10–30		3,520	34,000	50	32	57	6	0	0	2	3	0		
	2nd day		12–18	12–18	3,640	22,500	36	38	48	10	0	0	3	0	0		
442	10 every	10–30	10–30		3,100	30,000	54	36	62	1	0	0	1	0	0		
	2nd day		12– 1	12– 1	3,150	18,500	47	27	12	42	8	1	0	6	4	+	+
430	10 every	10–30	10–30		3,200	27,500	56	27	60	7	0	0	3	3	0		
	2nd day		11–28	11–28	1,800	13,000	37	38	18	16	0	0	1	6	21	+	++
425	10 every	10–30	10–30		2,780	29,500	53	23	65	5	0	0	3	4	0		
	2nd day		11–25	11–25	820	14,000	18	48	21	25	0	0	2	4	0	+	
429	10 every	10–30	10–30		2,990	30,500	53	27	70	2	0	0	0	1	0		
	2nd day		11–28	11–28*													
412	80	10–30	10–30		3,450	36,000	54	35	51	7	1	0	2	4	0		
	80	11– 1	11– 4	11– 4	3,260	34,000	56	29	51	15	0	0	2	3	0		
464	80	10–30	10–30		3,160	34,500	55	27	60	5	0	0	3	5	0		
	80	11– 1	11– 7	11– 7	3,180	32,500	55	25	58	12	0	0	2	3	0		
431	80	10–30	10–30		2,980	34,000	56	29	58	9	0	0	2	2	0		
	80	11– 1	11–10	11–10	3,000	34,000	52	34	40	20	1	1	2	2	0		
411	80	10–30	10–30		3,100	28,000	54	34	56	8	0	0	1	1	0		
	80	11– 1	11– 6	11– 6	2,980	30,000	57	32	58	7	0	0	0	3	0		
451	10 daily	12– 1	12– 1		3,280	26,000	57	24	62	10	0	0	2	2	0		
			12–30	12–30	3,000	14,500	48	43	32	19	0	0	3	3	0		

*Died of ruptured liver.

TABLE I—*Total and differential blood counts of birds experimentally affected with Crotalaria spectabilis Roth seed poisoning—Concluded.*

Bird	Seeds Fed	Date Fed	Date Blood Count	Date Autopsy	Erythrocytes	Leukocytes	Hemoglobin (Sahli)	Polymorphonuclears	Lymphocytes				Basophiles	Monocytes	Myelocytes	Polychrome Erythrocytes	Basophile Erythrocytes
									Mature	Vacuolar	Immature	Degenerative					
97-13	10 daily	12- 1	12- 1		3,170	30,000	54	36	54	6	0	0	1	3	0		
			1- 8	1- 8	2,870	33,500	42	44	14	14	0	0	0	28	0	+	++
91-3	10 daily	12- 1	12- 1		3,000	29,500	58	25	68	4	0	0	2	1	0		
			1-26	1-26	2,150	10,000	37	10	8	36	12	0	2	4	28	+	
91-8	10 daily	12- 1	12- 1		2,960	28,500	53	40	50	8	0	0	0	2	0		
			1-28	1-28	1,000	12,500	30	17	9	51	3	1	0	3	16	+	
471	10 daily	12- 1	12- 1		2,850	30,000	52	24	64	10	0	0	1	1	0		
			2- 5	2- 5	1,270	13,500	24	12	14	54	6	2	0	6	6	+	+
492	5 daily	11-15	11-15		3,120	29,500	54	20	71	5	0	0	2	2	0		
			12-30	12-30	2,430	25,000	45	22	20	43	0	0	1	14	0		
493	5 daily	11-15	11-15		2,980	27,000	56	27	63	8	0	0	2	0	0		
			1- 6	1- 6	1,870	20,000	30	14	21	43	0	1	0	7	14	+	++
478	5 daily	11-15	11-15		2,870	34,000	53	34	55	6	0	0	2	3	0		
			1-28	1-28	2,580	28,500	46	23	20	40	7	0	2	8	0	+	
405	5 daily	11-15	11-15		3,160	32,500	55	37	52	9	0	0	0	2	0		
			2-27	2-27	980	14,500	20	15	23	48	0	1	2	12	0	+	
417	5 daily	11-15	11-15		2,870	31,500	53	26	64	6	0	0	1	3	0		
			2-16	2-16	2,260	16,000	37	20	25	48	2	0	0	5	0		+
92-6	Control		11- 1		3,250	32,000	57	35	55	6	0	0	2	2	0		
			2- 7	2- 7	3,000	33,000	53	30	58	0	0	0	1	2	0		
432	Control		11- 1		3,150	31,500	56	27	60	7	0	0	2	4	0		
			2-7	2- 7	2,980	30,000	55	40	51	6	0	0	2	1	0		

ing. Considerable edema usually is observed in the stroma of the villi, more particularly at the extremities. Foci of histiocytes, often loosely arranged, frequently are observed at the base of the mucosa. These foci are often so extensive as to obliterate the glandular tissue and villi almost completely.

The epithelial lining of the gizzard, as well as the musculature of this organ, shows cloudy swelling. Cloudy swelling is present to a marked degree in the parenchymatous cells of the pancreas, in the isles of Langerhans, and in the glandular epithelium of the adrenal and thymus glands. Slight interstitial edema also is observed frequently in these organs.

The myocardium shows irregular cloudy swelling; in many areas the cells are practically devoid of cytoplasm. The myocardium is generally congested, is slightly edematous in some areas and occasionally shows small hemorrhages. The subendothelial tissue often shows edema.

In the more acute cases the liver shows intense passive congestion; in the chronic form passive congestion is not so marked but is still present in most cases. Pigmentation of the hepatic cells occurs in the liver of only one bird. The hepatic cells are generally in a state of cloudy swelling. In chronic cases the parenchyma of the organ appears as a structureless mass. Many of the hepatic cells lose much of their cytoplasm. Slight fatty degeneration is present in the liver of one bird. Many foci of necrosis occur, this lesion being usually more prevalent in and near those areas where passive congestion occurs. Hemorrhages in the parenchymatous tissue are common as are also areas of edema.

Edema also is present in a zone surrounding the interlobular blood-vessels. In such areas necrosis also is observed. The epithelium of the bile-ducts, as well as the muscle coat of the interlobular blood-vessels, shows considerable cloudy swelling. Irregular and loosely arranged foci of histiocytes often occur in the intralobular tissue and in the interlobular tissue, the latter usually in conjunction with the interlobular vessels. Occasionally few of these foci show proliferation. Slight edema often is observed in the interlobular tissue. The capsule of the liver usually is thickened and edematous and occasionally contains small hemorrhages. A hematoma occurs in the capsule of the liver of one bird. Slight edema and proliferation of connective tissue occur in some instances under the capsule.

The glomeruli often are enlarged and show considerable congestion. Occasional glomeruli show slight edema and degenera-

tion. The epithelial cells of the cortex show well advanced cloudy swelling. Much débris is present in the lumen of the tubules in many instances. Occasionally a tubule or group of tubules are almost completely denuded of epithelium. Many cells are often almost devoid of cytoplasm. A few small foci of proliferation of connective tissue and foci of histiocytes occur between the tubules. Much congestion often is evident in the cortex. However, congestion usually is more prevalent in the medulla. The cells of the tubules in the medulla also show cloudy swelling. There are a few intertubular foci of proliferation of connective tissue. An area surrounding the larger collecting ducts invariably shows edema.

Irregular areas of congestion and slight edema are present in the alveolar walls. Compensatory emphysema occurs to some extent in those areas least affected. The base of the prominences which contain the smooth muscle of the bronchioles is usually extensively edematous and shows deposits of pigment. The prominences themselves often show extensive involvement. A few scattered hemorrhages occur. Considerable alveolar epithelium shows cloudy swelling. Interlobular and perivascular edema frequently occur. Loosely arranged perivascular foci of histiocytes often are present.

Blood picture: In acute cases of poisoning the blood picture remains practically normal.

In the chronic form of poisoning many birds show a marked decrease in the total number of erythrocytes with a corresponding decrease in the hemoglobin reading (Sahli). The lowest erythrocyte count is 820,000 with a hemoglobin reading of 18. Most birds also show a considerable decrease in the total leukocyte count, the lowest being 10,000. The differential blood counts usually show a slight decrease in polymorphonuclear leukocytes, a marked decrease in mature lymphocytes, a similar increase in vacuolar lymphocytes, and an occasional increase in immature lymphocytes, monocytes and myelocytes. Polychrome and basophilic erythrocytes are present in the blood of many birds.

SUMMARY

Innumerable petechiae in the serous membranes and visceral fat as well as a dark liver, marbled in color, are the outstanding lesions in the acute type of *Crotalaria spectabilis* Roth poisoning in the domestic fowl. In the chronic type of poisoning necrotic enteritis, ascites and anemia are observed; the liver often appears as in the acute type of poisoning.

Passive congestion, edema, cloudy swelling and occasionally foci of necrosis in the parenchymatous tissues, smooth muscle and serous membranes dominate the histopathology.

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Horses and Mules

According to the annual survey made by the U. S. Bureau of Agricultural Economics, the number of horses on farms on January 1, 1937, was estimated at 11,527,000 head, a reduction of 108,000, or 0.9 per cent from the 1936 figure. This is the smallest decline in horses recorded in any year since the equine population began to decrease 20 years ago. The number of colts under one year of age was reported to be about 6 per cent larger than the 1936 figure and the largest since 1923.

The number of mules reported was 4,603,000, a reduction of 81,000 head, or 1.7 per cent less than the previous year. The number of mule colts under a year old was 20 per cent greater than a year ago and the largest since 1929.

These figures confirm forecasts made by the Horse and Mule Association of America. Horses are gradually approaching a condition of balance, where colts foaled will equal deaths. Mules will probably decline in total numbers for several years, because jacks are so scarce that the production of mule colts cannot be increased as rapidly as horse colts.

The average value of horses and mules is higher than the previous year. The greatest increase in prices has been among colts. A decrease in the value of other live stock was reported.

NEW UNION STATION AT OMAHA

A CULTURE MEDIUM FOR ERYSIPELOTHRIX RHUSIOPATHIAE*

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The scant growth of *Erysipelothrix rhusiopathiae* on all laboratory culture media hertofore recommended is one of the difficulties encountered during routine work with this microörganism.

Recently Deem[1] recommended yeast-tryptone broth as a favorable medium, especially for the preparation of agglutination antigens. According to our experience, the yeast-tryptone medium of Deem did not prove superior to the liver-digest solution of Meyer and Dubovsky.[2] The latter medium gives a positive test for tryptophane when properly prepared. The growth and density of cultures of *E. rhusiopathiae* grown in Deem's yeast-tryptone broth and liver-digest solution was approximately the same, which would indicate that the amino-acid tryptophane, rather than yeast, is desirable for growth of this microörganism. We have recently observed that liver digest solution enriched by the addition of 2 to 4 per cent sterile, normal horse serum proved superior to any medium heretofore recommended for *E. rhusiopathiae*.

The method of preparation is as follows:

Liver-digest solution (basal medium)
Bacto-peptone 1%
Sodium chloride 0.5%

Adjust pH to 7.6 or 7.7 before sterilization. Distribute in tubes or flasks and sterilize in the autoclave for 20 minutes at 15 pounds pressure. After sterilization, add 2 to 4 per cent of sterile, normal horse serum and incubate for 48 hours at 37° C. as a check on sterility.

Old stock strains as well as recently isolated strains of *E. rhusiopathiae* grow rapidly in this medium. It also proved excellent for the recovery of either *E. rhusiopathiae* or *Corynebacterium pyogenes* from polyarthritis lesions in lambs and swine. Broth cultures attain their maximum growth and turbidity in 24 hours at 37° C. without sedimentation. Gram-positive diphtheroid forms predominate at this time with occasional filamentous forms. The density ranges from 2.5 to 3 cm as measured with a Gates opacimeter.

Agar slants or plates may be prepared by dissolving Bacto-agar in the above described fluid media, 2 to 4 per cent sterile

*An abstract of this paper was presented at the Annual Conference of Research Workers in Animal Diseases, Chicago, Ill., December 1, 1936.

horse serum being added aseptically after sterilization. Colonies appear on serum-enriched, liver-digest agar after incubation for 24 hours, reaching their maximum size of 0.5 mm in about 48 hours, and are larger than on other media heretofore recommended. Rough and smooth colonies can be readily distinguished on plate cultures after incubation for 48 hours.

Twenty-four-hour, serumized, liver-digest cultures of *E. rhusiopathiae* can be used as whole culture antigen for the agglutination test by diluting with sterile liver-digest solution, pH 7.6, to a density of 5 cm (Gates scale) and the addition of 0.5 per cent phenol, the diluted and phenolized broth cultures being allowed to remain at room temperature for 24 hours before being used in the agglutination test.

Definite agglutination occurs with positive sera after incubation for eight hours at 37° C. and is complete in about 16 hours. Centrifugation of the agglutination tubes is not necessary, as agglutination is sharply defined and more easily read than is the case with some forms of agglutination antigen heretofore recommended.

Comparison was made using saline suspensions of *E. rhusiopathiae*, alcohol-treated antigen of Deem, and the whole culture antigen described. Commercial anti-swine erysipelas serum and positive sera from sheep convalescent from erysipelas infection agglutinated all of the antigens in the same dilutions.

Although whole culture *E. rhusiopathiae* antigens do not remain sensitive longer than three or four days after preparation, they have the advantage of being prepared quickly from routine culture material in the quantity desired. In our experience they are as sensitive as suspensions of the organism in physiological sodium chloride solution or the alcohol-washed antigen of Deem. The whole culture antigen does not spontaneously agglutinate during the incubation period. The diluted, whole-culture antigen can also be used without addition of phenol, but it is not advisable due to the possibility of infection among laboratory personnel. Formalinized antigens did not prove satisfactory, as antigens so treated were not completely agglutinated by known positive sera.

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Teacher at Mont Alto, Pa., asked Robert to name the sexes. He replied, "Male sex, female sex and insects."

Animaldom.

A COMPARISON OF THE TUBE AND PLATE METHODS OF TESTING FOR BANG'S DISEASE IN ELK*

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Bang's disease has previously been reported in elk. Tunnicliff and Marsh,[1] in 1931 and 1932, tested 105 elk which had been running on a range with buffalo and found 8 per cent reactors. They found no reactors in another group of 45 elk, which were tested in 1931 and 1932 and which had not been running on a range where buffalo ran. Rush[2] reported three reactors out of 32 elk tested in a herd that wintered near where buffalo wintered and no reactors were found out of 35 tested from the vicinity of Mammoth, 30 miles from a buffalo range.

In November, 1935, eleven blood samples from elk were received from the State Veterinarian of Wyoming, for routine testing for Bang's disease. These samples were tested by the plate agglutination test, with a rapid commercial antigen which had been giving reasonable agreement with the tube test. They were all negative in the 1:50 and higher dilutions. Because these samples were from elk, the results were interesting. The samples were then tested by the official agglutination tube test. To this test two samples were strong reactors, being positive in dilutions of 1:50, 1:100, 1:200 and 1:400. Another sample was called a suspect, being positive in the 1:50 dilution and incomplete at 1:100. The plate and tube tests were repeated with the same results as obtained on the first tests.

This percentage of disagreement by the tube test in the eleven samples taken at random was much higher than the authors had been finding in cattle with the plate antigen used. This stimulated interest in this phase and so more samples were secured from elk.

There were 132 more elk samples obtained which were suitable for testing. They were obtained in December, 1935, from elk which were slaughtered in the herd reduction program. These were all tested by the official tube test and with two commercial plate antigens which had given us reasonable agreement with the tube test in cattle. The results are recorded in table I. Those which gave complete agglutination in the 1:100 dilution or higher were called reactors, and suspects were those showing complete or incomplete agglutination in the 1:50 dilution. There

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TABLE I—*Wyoming elk tested for Bang's disease, December, 1935.*

METHOD AND ANTIGEN USED	TOTAL TESTED WITH ALL ANTIGENS	REACTORS		SUSPECTS		NEGATIVE	
		No.	%	No.	%	No.	%
Official tube test	143	28	19.6	17	11.8	98	68.6
Plate antigen 1	143	19	13.2	14	9.8	110	76.9
Plate antigen 2	143	22	15.4	11	7.6	110	76.9

Reactor=Complete agglutination 1:100 or higher.
Suspect=Complete or incomplete 1:50 dilution.
Note: Plate antigens used were two of the most efficient commercial ones on the market which give reasonable agreement with the tube test in cattle.

were 28 reactors (19 per cent) out of the 143 tested by the tube test. There were 19 (13 per cent) reactors to the plate test with antigen 1 and there were 22 reactors (15 per cent) with plate antigen 2. There were 17 suspects to the tube test, 14 to the plate test with antigen 1 and eleven with plate antigen 2.

Failure to get reasonable agreement between tube and plate tests with one or both antigens was encountered in 19 of the 143 samples tested. This was over 13 per cent. Fifteen of these 19 samples are tabulated in table II. It will be seen that strong reactors to the tube test were negative to the plate test. Also that in some samples strongly positive to the tube test, plate antigen 2 gave positive results while plate antigen 1 gave negative or suspicious results. On the other hand, sample 49 was positive in dilutions of 1:200 by the tube test and with plate antigen 1 whereas plate antigen 2 gave a positive reaction in only the 1:50 dilution.

The four other samples of the 19 mentioned above which were strong reactors to the tube test were tested with four plate antigens which gave reasonable agreement with the tube test in cattle. The results are tabulated in table III. It is evident that none of these plate antigens was nearly so satisfactory in detecting reactors as the tube test.

The elements of time and temperature in testing by the plate test were also worked upon. It was found that ten minutes was necessary many times in getting a sample which was positive to the tube test to react to the plate test. All the readings for the plate tests reported were read at the end of ten minutes.

It can be seen that the 19 per cent infection found and reported here on 143 samples taken at random is much higher than has been reported by others in 1931 and 1932. Also, that the tube test with a 72-hour incubation and a polyvalent antigent was

TABLE II—*Results of tests on 15 samples in which reasonable agreement was not obtained with tube and plate tests.*

SAMPLE	METHOD AND ANTIGEN	DILUTIONS			
		1:50	1:100	1:200	1:400
115	Official tube	+	+	+	+
	Plate antigen 1	+	1	—	—
	Plate antigen 2	+	1	—	—
36	Official tube	+	+	+	+
	Plate antigen 1	+	1	—	—
	Plate antigen 2	+	+	+	—
49	Official tube	+	+	+	—
	Plate antigen 1	+	+	+	—
	Plate antigen 2	+	—	—	—
108	Official tube	+	+	+	+
	Plate antigen 1	+	1	—	—
	Plate antigen 2	+	+	+	—
106	Official tube	+	+	1	—
	Plate antigen 1	+	—	—	—
	Plate antigen 2	+	1	—	—
104, 125	Official tube	+	1	—	—
	Plate antigen 1	1	—	—	—
	Plate antigen 2	—	—	—	—
26, 51, 83	Official tube	+	1	—	—
	Plate antigen 1	—	—	—	—
	Plate antigen 2	—	—	—	—
3, 116, 136	Official tube	+	1	—	—
	Plate antigen 1	—	—	—	—
	Plate antigen 2	—	—	—	—
A	Official tube	+	+	+	+
	Plate antigen 1	—	—	—	—
B	Official tube	+	+	1	—
	Plate antigen 1	—	—	—	—

TABLE III—*Comparison of tube and plate methods used in testing four samples.*

SAMPLE	METHOD AND ANTIGEN	DILUTIONS			
		1:50	1:100	1:200	1:400
122	Official tube	+	+	+	1
	Plate antigen 1	+	+	1	—
	Plate antigen 2	+	+	1	—
	Plate antigen 3	1	—	—	—
	Plate antigen 4	—	—	—	—
3	Official tube	+	+	+	+
	Plate antigen 1	—	—	—	—
	Plate antigen 2	+	+	1	—
	Plate antigen 3	+	+	—	—
	Plate antigen 4	—	—	—	—
157	Official tube	+	+	+	+
	Plate antigen 1	+	+	—	—
	Plate antigen 2	+	1	—	—
	Plate antigen 3	+	—	—	—
	Plate antigen 4	+	+	—	—
45	Official tube	+	+	+	+
	Plate antigen 1	+	+	—	—
	Plate antigen 2	+	+	+	+
	Plate antigen 3	+	+	+	—
	Plate antigen 4	+	+	1	—

found to be considerably more accurate than the plate or rapid test.

ACKNOWLEDGMENT

The authors wish to thank Dr. H. D. Port, State Veterinarian of Wyoming, for furnishing the samples tested.

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Unusual Accident

A man was killed recently near Epworth, Iowa, when struck by an automobile driven by an undertaker. The accident occurred while the victim was attempting to rescue an injured dog from the highway.

Fortune is like glass; when she shines, she is broken.—Syrus.

UNDEVELOPED GROUNDWORK*

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American Veterinary Medical Association

Many years of study and experience were required to plot a workable setup for the American Veterinary Medical Association; to be more exact, more than seventy years. Its geographic jurisdiction is vast, its sectional problems varied, and the current personal interests of its members are diversified, if not in some respects antagonistic, to mention but a few of the more baffling obstacles strewn along its path. Its predicaments are much the same as those of any American organization that has aspired to nation-wide affluence. Profiting by the experiences of national associations in other fields, it was long since realized that membership organizations in the different states and provinces amalgamated into a single unit would be required to further the projects of the profession and plans to this end were studied and discussed unceasingly by its administrative councils during the past thirty years.

The appointment of a resident secretary in each of the states, provinces and territories was the first step taken in that direction. They were the Association's local representatives charged with the procurement of new members and general promotional work. As I recall the situation when this step was taken, state associations were small, non-existent, none too viable and, regrettably, not hopeful units for a nation-wide amalgamation such as was recently accomplished. With membership small and financial support lacking there was nothing to do but to carry on with precept and example until greater interest in association work developed in the states. When this moment arrived, several years ago, the state organizations were invited to participate by becoming member associations and to send delegates to form a House of Representatives, or, in other words, to take one more forward step toward a more complete federation of interests.

As the general objective is to bring every eligible veterinarian into the state organization and through it into the national body, it is evident that much remains to be accomplished, encouraging as the prospects now appear. There is still considerable groundwork to be done. It is remarkable how few members of the

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national association, how few officers of state associations, how few eligible members, understand the makeup of the national organization of the present time. In the quest for facts in this respect, one becomes astounded at the insufficient interest members of the veterinary profession take in matters of such outstanding importance to them. "Undeveloped Groundwork" is therefore a rational title for this paper.

Neglected Opportunities

The American veterinarian has not only been delinquent in seizing upon association work as a means of helping himself but also in overlooking such work as the one and only way of doing so. To those who devote considerable thought to the situation, it is plain that our most grievous troubles all trace to this single cause. In passing, it might be mentioned to advantage that the groups who have participated eagerly in association work have given too little thought to the main professional questions. That is to say, programs abound with theoretical and clinical subjects aiming to edify the membership in matters of individual interest but omit the professional subjects required to keep informed on the ways and means of developing the profession as a whole. The consequence of the omission has been unfortunate and needs attention.

We know too little of the profession's plans, methods and aims and we give too miserly encouragement to the leaders who volunteer to serve for us. The veterinary profession of this country has never lacked leaders but it has lacked folks to lead, in numbers and kind. There is no space here to quote statistics on the ratio of total eligible veterinarians to total association memberships except to point out that the ratio is not complimentary and that it tells the whole story of our unsatisfactory affluence in the domain of animal production. In short, the veterinary profession does not have a ghost of a chance of advancing beyond the ambitions exemplified by association membership, but the reasons are too numerous to dwell upon here.

The veterinary circle in recent years has been stirred with the notion that the public should be enlightened about its work and its importance to mankind. This trend is a veritable obsession which means the possession of one idea to the exclusion of others, and it has taken form in policy committees, public relations committees, publicity programs and other devices charged with drawing popular attention to the veterinary service. Rational and commendable as these agenda are under the

circumstances which have been allowed to develop, careful surveys of the situation carried out to get at causes and effects all lead to the conclusion that the veterinarian himself needs edification also on the application of his art to the best advantage in welfare work. In that respect we are "non-graduates."

THE ASSOCIATION SCHOOL

Too few have attended the association school, and too few associations have had professional subjects in their curricula. Reform measures not based upon events of the past seldom amount to much and those not grounded upon the facts of the present are apt to fail. What I mean to say is that we should know more about ourselves and the operation of our profession and that the associations are the only school where plans can be laid and information furnished for guidance. Those who have remained skeptical about the benefits of associations seem to forget that but for even our insufficient organization their profession would still be an unnamed trade trekking sickly along under the common law.

Anyone curious enough to look into the matter will agree that the advance of the veterinary profession has corresponded precisely to the advance of the associations. The work of no branch of veterinary medicine has ever lagged behind nor stepped ahead of them. As a matter of fact, whatever your associations are so are you. Assuming this to be true, the main task confronting the American veterinarian is to wake up and flock to organized veterinary medicine and thus create the only agency capable of clearing away the obstacles that clutter the lane of the unorganized. The American Veterinary Medical Association and the state associations which are now components provide that agency. For the veterinary profession to develop a future more nearly consonant to the knowledge of medicine than the past has been and the preesnt is, association membership must first be brought up to a more reasonable percentage of total eligibles. 'Til then we shall probably continue to argue over details among ourselves and go on drifting to who knows where.

The development of organized veterinary medicine is in your hands. The national association is now strictly democratic. The territory is divided into ten districts, each represented on the Executive Board by a member elected by postal ballots. Your district, for example, comprises Wisconsin, Indiana and Illinois, and the members of the Association from these three states have selected me as their agent on the Executive Board and I am here

to impress upon you the importance of seizing the opportunity of exercising your rights in making the selection, for here is a body of executives, each serving for five years, who hold long sessions twice a year to administer the affairs of the organized profession and to pilot its ship. If this elected board seems conservative or perhaps reactionary, it is because the passenger list is small and those aboard are undecided as to the course to chart. It needs the council of a larger and better informed crowd which can be furnished only through association memberships.

Present Arrangement Satisfactory

In addition to the ten members of the Board elected by postal ballots, a member-at-large is elected every five years and the President and the President-elect are *ex-officio* members with power to vote. Inasmuch as these three members are elected by the members present at meetings, the democratic character of this administrative body is self-evident. As ten members of the Board are selected by the association members in their respective districts and three by the association members in attendance at the annual meetings, the arrangement is certainly satisfactory.

But, it is through the instrumentality of the House of Representatives that every eligible veterinarian in the country obtains a voice in the operations of the national association. Each member association chooses its representative to serve two years and the voting power of each varies with the number of national association members his state has on the roll. Obviously, therefore, a state with a small membership is not so well represented as one with a larger membership, notwithstanding that the former may be equally important from the standpoint of its veterinary service. It is, therefore, within the power of the small-membership states to increase their representation by doing what I am here to recommend, namely, join the national association and thus take a step nearer to the day when practically all eligible veterinarians will have to join the national through the state organization.

It is obvious that such a radical step would not be feasible at the present time, desirable as it is to accomplish that objective as soon as possible. At the present time, one is justified in pronouncing the American Veterinary Medical Association a truly representative organization, starting out to a brilliant future. A practical means has been found to dovetail the affairs of the veterinary profession of a vast agricultural nation into a solid unit, in other words, a means of providing universal franchise

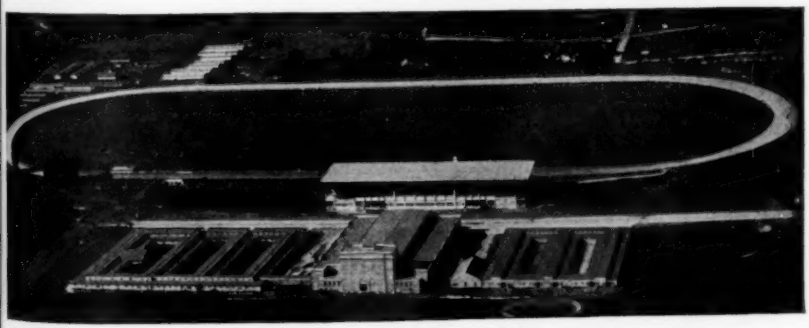
to eligible veterinarians. So, acquire the right to vote on your own fate.

In closing I wish to leave these recommendations: Join the American Veterinary Medical Association, interest yourselves in the election of the Executive Board member of your district, see that your association is properly represented in the House of Representatives, expand the membership of your association, and last, but not least, include papers and discussions on professional questions in your programs.

Michigan Community Health Project

The Michigan Community Health project, sponsored by the W. K. Kellogg Foundation, of Battle Creek, held a two-day conference of the veterinarians from seven counties, in Battle Creek, on February 15-16, 1937. The conference was in the nature of a post-graduate course in subjects dealing with health problems in which the veterinarian is vitally interested.

The speakers on the program included Dr. Ward Giltner, Michigan State College, who discussed "Undulant Fever"; Dr. Warren P. S. Hall, Health Department, Toledo, Ohio, who discussed "Meat Inspection," and Dr. S. D. Johnson, of Cornell University, Ithaca, N. Y., who talked on "Mastitis." Dr. John K. Glover, Indianapolis, Ind., spoke on "Biological Products in the Control of Animal Diseases," and Dr. R. B. Harkness, who is in charge of the health departments of Barry and Eaton counties, discussed the subject, "Veterinary Participation Program," in its relation to the work of the W. K. Kellogg Foundation.



AK-SAR-BEN COLISEUM, WITH RACE TRACK AND EXHIBITION GROUNDS, OMAHA

TREATMENT OF IMPACTION IN THE HORSE WITH LENTIN, THE NEW RAPID-ACTING CATHARTIC*

By M. V. BURGETT, *East Chicago, Ind.*
and G. W. BARDENS, *Lowell, Ind.*

The first step leading toward the synthesis of lentin and other therapeutically valuable choline derivatives was made as far back as 1849. During that year, Strecker,[1] a German chemist, isolated a platinichloride base from pig bile. That he had little or no premonition of the importance of his discovery is indicated by the fact that he did not report further on the subject for 13 years, or until 1862, when he published the formula, a further description, and named the base choline.[2]

In the meantime, in 1852, Von Babo and Hirschbrunn,[3] by hydrolysis of the alkaloid sinapin from white mustard seeds, had prepared a strong base which was well characterized by its platinichloride. It was named sinkalin (from sinapin and alkali). The identity of this base from mustard with that from pig bile was established by Claus and Keese,[4] in 1867, but nevertheless choline, Strecker's (later) name passed into general use.

In 1865, Liebrich[5] obtained a base by hydrolysis of the brain substance, protagon, and termed it neurin. The identity of Liebrich's protagon base with choline was established by Dybkowski[6] in 1867.

These preliminary discoveries furnished the initial impetus which started work on choline in every scientific center. Strecker, Von Babo and Hirschbrunn, Claus and Keese, Liebrich, and Dybkowski were followed by Schulze,[7] Trier,[8] Kinoshita,[9] and numerous other investigators who, through the succeeding decades, collectively isolated choline in small amounts from practically all the organs and tissues of the body, and from the seeds, roots and bark of many plants. In 1914, Barger,[10] basing his assertion on the fact that choline had been discovered to be a constituent of lecithin, said that it is probably a constituent of all living cells. The amount of choline from most sources is very small, running usually about 0.02 per cent in animal viscera and seeds. Egg yolk is the most convenient natural source.

As this work progressed, year after year, decade after decade, and the widespread distribution of the chemical was more and more obvious, it became apparent that the rôle of choline in nature must have some important significance. But the problem was not easily solved.

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The study of choline was greatly facilitated, particularly from the therapeutic aspect, almost from the first, by the synthesis of the chemical by Wurz,[11] in 1867, by the action of trimethylamine on ethylene oxide in concentrated aqueous solution. This made the chemical available in sufficient amounts for therapeutic experimentation. It also made possible the synthesis of choline compounds and derivatives.

The importance of this is that while choline has a therapeutic action, it is weak and evanescent compared to the powerful actions exhibited by many of its derivatives. Thus synthesis opened the way for therapeutic experimentation with choline and also with a vast number of compounds and derivatives based on choline.

In 1911, Hunt and Taveau[12] reported study of the action of a large number of synthetic choline derivatives. And in particular, acetylcholine, $(CH_3)_3N(OH)CH_2CH_2O.OC.CH_3$, which was reported as remarkable in being 100,000 times as active as choline itself.

And so it was that many investigators, indeed, shared in the long, tedious 80 years of effort that culminated in the work of Sir Henry Dale,[13] in London, in 1929. His discovery that acetylcholine could be increased in amount in the body tissues by vague stimulation, particularly in the bowel musculature, furnished the clue which led to its identification as a body control chemical.

It is now known that acetylcholine is a natural body chemical formed at the myoneural junctions of the parasympathetic nerves, and that it serves as a chemical mediator for distribution of parasympathetic nervous impulses to the individual cells. This action places it as the physiological antagonist to epinephrine, which is the mediator for the opposed or sympathetic side of the important autonomic nervous system.

The action of acetylcholine being, therefore, exactly opposed to epinephrine, its administration, and particularly the administration of some of its derivatives, results in constriction of the bronchioles, slowing of the heart, lowering of blood pressure, dilatation of peripheral blood-vessels, increase of glandular secretion of tone of smooth muscle generally, particularly of bladder and bowel. In each instance, epinephrine does the opposite; and these two body chemicals, when formed in proper amounts, are responsible for the fine balance between the two divisions of the autonomic nervous system—sympathetic and parasympathetic—essential to health.

Sir Henry Dale, in 1929, in connection with the work which resulted in the delineation of the exact rôle of acetylcholine in

the body, was able to show conclusively that acetylcholine is produced at the myoneural junctions of the parasympathetic nerves. He recovered it from the muscles in which it had been liberated. Acetylcholine, in the form of acetylcholine chloride, came into therapeutic use. Its administration produces the characteristic phenomena of parasympathetic stimulation; but its therapeutic value is interfered with because of rapid destruction in the organism by a tissue and blood esterase. In the search for more stable choline compounds, a large number of choline derivatives were synthesized, and attempts to evaluate each of them therapeutically followed.

It was learned in the course of this work that the various synthetic radical substitution compounds of choline vary considerably in their properties. It was also found that there is a tendency for the compounds to have selective actions on different tissues and organs. One synthesized by Major and Cline,[14] acetyl beta methylcholine chloride, for instance, has been used in human medicine. It is now widely used by physicians in the treatment of paroxysmal tachycardia and peripheral vascular diseases.

On account of the high incidence of digestive disturbances among domesticated animals, it was thought that lentin, a choline derivative which was discovered to expend its action more preponderantly on the alimentary canal, should be the one of greatest value in the veterinary field. This has been borne out by the results reported on the use of the chemical by many veterinarians, particularly in Germany, where it was first introduced.

Lentin, or carbaminoxyl-choline chloride, structurally may be represented as $NH_2.CO.OCH_2.N(CH_3)_3Cl$. It belongs to the group of carbamic acid esters of choline which, because of unusual stability and very slow destruction in the organism, exhibits an extraordinary potency, particularly on the gastro-intestinal tract. Besides its potent action, which is many times that of choline or acetylcholine, it has additional advantages not enjoyed by choline derivatives generally which make it of particular utility in veterinary medicine. It is stable both as a solid and in solution, it is thermostabile and can be sterilized by boiling, it is effective orally without the necessity of excess administration, it is effective by easy subcutaneous injection—not requiring intravenous administration, and it is cheap.

Lentin, first introduced into veterinary practice in 1932, was tried in several diverse live stock diseases wherein parasympathetic stimulation is indicated, and with considerable success in many of the conditions. However, as in this paper we are con-

cerned wholly with the therapeutic effects of the chemical in colic and impaction in the horse, references and quotations from the published papers of German veterinarians and others will here be confined to their comments concerning its effects in the aforementioned conditions. The bibliography will facilitate easy reference to the complete publications if desired.

Foreign veterinary journals during the past several years have carried many papers on lentin. The very large number of these reports precludes any reference here to all or even to any large proportion of them. Suffice it to say that these publications have one and all been highly favorable to lentin. It is now in use by the majority of German veterinarians; and has been adopted by the German army as standard equipment to replace arecoline, eserine, and barium chloride, the rapid-acting cathartics formerly used. As examples of opinions concerning lentin, excerpts from a few of the papers mentioned above will be quoted here. Nöll[15] states:

> The effect of lentin on peristalsis, salivation, and secretion of gastric juice is extremely pronounced.

Velten[16] writes:

> The stimulating effect of lentin on the isolated stomach of the dog was five times that of arecoline, and it showed a much stronger stimulating effect on the stomach muscles of the living dog and on the secretion of its gastric juices. It stimulates the gastric tonus and contraction in frogs, rats and cats with simultaneous increase of gastric secretion.

K. Neuman-Kleinpaul and Rescher[17] report:

> Lentin is an active remedy in cases of obstruction of the cecum and colon in horses, and it quickly cures tympanites.

E. Haertle[18] writes:

> Lentin is of service in veterinary practice because of its rapid and sure effects on the stomach and intestines, because it has no secondary effects on the heart, because it may be given by easy subcutaneous injection, and because of its cheapness.

Jacob[19] states:

> Lentin undoubtedly possesses a stimulating effect on the motility of the stomach and intestines and on general gland secretion as a consequence of stimulation of the parasympathetic nervous system. Its indications are those of arecoline, though the effect is more pronounced and it can be used safely by subcutaneous injection in patients with weakened hearts where arecoline is contraindicated.

H. Doeneke[20] reports:

> Lentin is better than arecoline and will probably replace it in the veterinarian's armamentarium. It stimulates glandular secretion in the whole of the alimentary tract better than any other known remedy. It has been established that, after lentin administration, the stomach and intestines produce about five times more digestive juice than after arecoline, the amount being eight times

more than in untreated animals (*i.e.*, arecoline increases the secretions 1 3/5 over normal, and lentin eight times over normal) . . . and lentin raises the tone of smooth muscle, increasing peristalsis.

Opperman[21] states:

> Subcutaneous doses of lentin of 3 to 5 mg, after the course of three hours, definitely soften the fecal masses of obstipation, especially in the large intestines. Its stimulating effect on gastro-intestinal secretions and peristalsis may last for as much as four hours. In slight obstructions, the impacted mass may be moved on by a single dose. If this does not occur satisfactorily, another dose of the same size should be given after four hours, and not delayed in any event more than eight to ten hours. The weariness which usually appears after the cessation of the action of lentin disappears in one to 1½ hours, and normal appetite reappears. Lentin brings about an abundant passage of fluid intestinal contents; but, since it does not increase thirst, the feces are no longer fluid after an hour from the peak of the action, but become mushy or loosely formed. While arecoline, eserine, and barium chloride strongly depress the heart, and should not be used in animals with weakened hearts, lentin can be used without harm.

These excerpts are from the publications of veterinarians who have used lentin extensively. They and many other veterinarians who have published reports on lentin agree that the chemical has an action that is peculiarly valuable in the treatment of digestive disturbances, particularly in impactions of the large bowel of horses. It is not only superior as a stimulant of peristalsis, but also increases secretions in such volume as to exert the greatly-to-be-desired effect of softening up the impacted masses. This combined action, together with the freedom of lentin from untoward effects on the heart and bowel (no heart failure and no superpurgation from lentin) are responsible for its popularity.

In order to test the value of lentin one of us (G.W.B.) treated a series of 21 cases with the following results:

Case 1: A 16-year-old farm work horse. Would not eat at noon. Had colic at 4 p. m. At 5 p. m. pumped stomach out, and pumped in a pint of oil with antiferments. Injected subcutaneously a 4-cc ampoule of lentin. Salivation and an increase in peristalsis started in a few minutes. Peristalsis increased rapidly, and there was a good evacuation in twelve minutes. Recovery was prompt and complete without further treatment.

Case 2: An eight-year-old gelding. Was retching severely and, according to the history, had been choked for several hours. To test the reputed value of lentin in this condition, injected one 4-cc ampoule subcutaneously. In twelve or 15 minutes, salivation started and in a little while became profuse with the animal swallowing repeatedly. There was complete recovery without further treatment.

Case 3: Registered mare, weighing 1,740 pounds. Had been ill ten hours when first seen. Getting up and down, rolling, and in great distress. Unknown treatment by another veterinarian to which animal had not responded. Passed a stomach-tube; no return except clear water. Gave pint of oil with antiferments and an ounce of chloral-hydrate. Injected 4-cc ampoule of lentin subcutaneously. In 20 minutes after giving the lentin, there was profuse salivation and a marked increase in peristalsis. Upon second call, made four hours later, found

animal fairly well recovered, but still in some pain. Gave second 4-cc dose of lentin. This caused profuse salivation in 15 minutes, and a marked increase in peristalsis which resulted in a bowel evacuation in 15 minutes, and a second evacuation in 45 minutes. The animal recovered without further treatment.

Case 4: An eight-year-old gelding. Ate half-pail of moldy silage. In violent pain; up and down, throwing himself, rolling. Diagnosis—impaction with fermentation and exogenic toxicosis. For the purpose of possible neutralization of toxins, gave a mixture of sodium nitrate, sodium thiosulfate and methylene blue, together with 250 cc of calcium gluconate solution, intravenously. Pumped stomach out and gave quart of oil with antiferments. No lentin given at this time. Four hours later, upon return call, found animal in still more violent agony; up and down and wild with pain. The nostrils were distended, pupils dilated, pulse rapid, heart throbbing, conjunctivae congested, and the temperature starting up. Little peristalsis and no bowel movements since previous call. Pumped stomach out and gave pint each of castor and mineral oil with antiferments. Gave another dose intravenously of the mixture of sodium nitrate, sodium thiosulfate and methylene blue. Merely as an experiment in this desperate case, gave 3 cc of lentin. On third call, four hours later, gave 16 ounces of a 1 per cent solution of mercurochrome intravenously. Gave one-half ounce of chloral, and animal seemed easier for a time. Gave 3 cc more of lentin. Next morning, seven hours later, the temperature had risen to 104° F. Pain was gone and animal standing still. Pumped stomach out, and gave another intravenous injection of the sodium nitrate, sodium thiosulfate and methylene blue mixture. The animal died several hours later. Consider case was too close to inflammation of the bowels at the start of medication for successful treatment.

Case 5: A nine-year-old mare, weighing 1,300 pounds. Owner found the animal ill in evening. Stall kicked down, and apparently had been ill most of day. In continuous pain and constantly getting up and down, wet with sweat, nostrils distended, conjunctivae congested, almost no peristalsis but no bloating. Diagnosis—impaction of large bowel. Pumped stomach out; no return except clear water and mucus. Gave pint of mineral oil with antiferments and barium chloride, 90 grains, with 15 grains of aloin. Then injected 4 cc of lentin subcutaneously. In a few minutes—not more than ten—there was profuse salivation and a marked increase in peristalsis, causing considerable distress. After a little while, a good peristalsis was built up by the lentin as it was much too soon for the barium chloride to act. On return call next morning, found animal still sick but with not so much pain and with a lot of peristalsis. Gave a pint of mineral oil with antiferments. Then injected 3 cc of lentin subcutaneously. This caused profuse salivation and an almost immediate increase in peristalsis, resulting in a bowel movement in two hours. The animal recovered without further treatment.

Case 6: A nine-year-old gelding. Appeared to be uneasy and in some pain, but no getting up and down, rolling, or straining to defecate. Would look first at one side and then the other. Rectal examination revealed an impaction of the rectum. Removed this manually, then injected 3 cc of lentin subcutaneously. This caused profuse salivation and a marked increase in peristalsis in a few minutes. During the following hour, there were two good bowel movements. Left oil to be injected into the rectum every few hours. The animal recovered without further treatment.

Case 7: Young gelding. Showed evidence of dull pain. Lying flat on side most of time with occasional look at one side or the other. Markedly diminished peristalsis. Diagnosis—impaction. Rectal examination confirmed this, revealing impaction of both colon and cecum.

No return with stomach-tube except clear water and mucus. Pumped in 1 pound of Epsom salt in 10 quarts of water with antiferments. Gave 4 cc of lentin subcutaneously, but could not stay to see its action. Left quart of mineral oil to be given in two doses, six hours apart. This was not needed, however. Owner reported later that in three hours the animal had recovered and its bowels were moving satisfactorily.

Cases 8, 9, 10 and 11: These four animals all had severe forage poisoning with toxemia and meningitis. All died in spite of rapid purgation, forage poisoning antidotes, and other treatment. Lentin was used on all four cases as a rapid-acting cathartic to sweep out the toxic material that had been ingested as quickly as possible to avert further absorption. This caused salivation and purgation started in each case in 20 to 30 minutes after the administration of lentin. Its action was all that could be desired in these four cases—a nicer purgative effect could not be asked for.

Case 12: A seven-year-old gelding. Sick an indeterminate time. Showed evidence of dull pains. If standing, would look first at one side and then the other; but would lie stretched out flat on side by the hour. Rectal examination disclosed an unusually dense impaction of the colon. Gave aloin 180 grains with correctives, 2 quarts of mineral oil, and one-half ounce of chloralhydrate. Then injected subcutaneously 4 cc of lentin. It promptly produced profuse salivation but there was no detectable immediate increase in peristalsis; impaction probably too dry. Left 2 quarts of mineral oil, a pint to be given every four hours. Felt very dubious about the prognosis. Next day, found there had been no bowel movement, and on rectal examination found the colon still very tightly impacted. Passed the stomach-tube and gave pint of mineral oil. Gave mercurochrome intravenously. Left oil, 8 ounces to be given every three hours. Felt case hopeless. Gave 3 cc of lentin subcutaneously—as an experiment. It caused salivation and some straining to defecate, but no peristalsis. Animal developed a temperature of 105° F. 16 hours later, and died two hours after that of inflammation of the bowels. Case probably too close to inflammation of the bowels before treatment started.

Case 13: An 18-year-old mare. Sick 20 hours before seen. At the farm on other business and owner wanted her looked at. He thought she would die and said he wouldn't pay a dollar for her treatment. Found her in violent pain, up and down, walking in circles, rolling. Pulse fast and peristalsis greatly suppressed. Diagnosis—impaction of small intestine. Decided to treat her on my own as an experiment with lentin. Gave 1 ounce of chloral orally and 4 cc of lentin subcutaneously; no other treatment at all. Her skin was dry, but shortly after the administration of lentin she started to sweat profusely, the water just dripping off of her. She salivated profusely, too. The owner was watching with his eyes "popping out" as I told him I was giving her the "kill or cure" treatment. In 20 minutes after giving the lentin, there was a marked increase in peristalsis. In an hour, the animal had four bowel evacuations and was entirely free from pain. This was a fine test of lentin and a gratifying recovery. The owner changed his mind about not paying for the treatment.

Case 14: A twelve-year-old mare. In poor condition with staring coat, and had passed some worms. Stomach and large bowel o.k., but there was violent pain and considerable suppression of peristalsis. Diagnosis—impaction of small intestine with worms. Gave 8 ounces of mineral oil and 20 grains of barium chloride. Then injected 3 cc of lentin subcutaneously. There was some peristalsis, but lentin increased it in a few minutes. In 20 minutes, there was profuse salivation and sweating and a marked increase in peristalsis with colicky pains, rapid breathing, dilated nostrils. The first bowel move-

ment occurred 1½ hours after the administration of lentin, and shortly afterward the pain ended. Next day, the animal passed two quarts of worms.

Case 15: An eight-year-old mare, weighing 1,600 pounds. Colic with violent pain, up and down, rolling, suppressed peristalsis. Diagnosis—impaction with fermentation. Gave aloin, 180 grains, and two Reeks' colic capsules. Injected 4 cc lentin subcutaneously. Lentin worked quickly, and at first aggravated the pain somewhat. It quickly induced free salivation and some sweating, and in 20 minutes had started up good peristalsis. The first bowel movement occurred in about 25 minutes and the animal recovered without further treatment.

Case 16: Same animal as case 15 above. Colic recurred a month later. Was found ill in the barn, in violent pain, up and down, trying to roll. Diagnosis—impaction with fermentation. Injected 4 cc of lentin subcutaneously, and it produced the same excellent results as before—salivation, sweating, and a rapidly increasing peristalsis that resulted in a bowel movement in less than an hour, ending the trouble.

Case 17: Heavy farm gelding. Found lathering with sweat, lame, and in great pain. Diagnosis—azoturia. Gave 1½ ounces of chloral, and put the animal down. Gave aloin, 180 grains, and 4 cc of lentin subcutaneously, the latter for quick purgative effect. It caused two good bowel movements, one in 30 minutes, and another in 1½ hours. Animal got up in four hours. Got good action from aloin next day. Animal was lame for four days and gradually recovered. The action of lentin in this case in producing quick purgation was all that could be desired.

Case 18: A wild, six-year-old broncho that had never had a halter on. Got into rye grain and had eaten enough to give him violent colic. Roped him and injected 4 cc of lentin subcutaneously. Was so wild did not attempt to give anything else, and this made it a good test for lentin. In about 20 minutes, the animal commenced to salivate profusely and sweat. The saliva ran down from his mouth in foamy strings and the animal was so wild he would strike at them with his fore feet, then he would run a while and stop and strike at the strings of saliva some more. There were three good bowel movements in three hours. In 2½ hours, a half-hour before the third bowel movement, the pain was all gone and he was quiet, unless one went near him. All the credit for this splendid recovery goes to lentin, as it was the only medicament given in this case.

Case 19: A six-year-old gelding, weighing 1,200 pounds. Dull pains, up and down, and part of time stretched out flat. Rectal examination disclosed impaction of the colon. Passed stomach-tube and gave oil and antiferments. Injected 3 cc of lentin subcutaneously. This caused salivation but no immediate increase in peristalsis was detectable. Seen again in five hours. Seemed worse; lying flat most of time. Passed stomach-tube and gave a pint of oil, antiferments, and one-half ounce of chloral. Then injected 2 cc of lentin. This caused salivation and a good increase in peristalsis, which was audible upon auscultation. Left oil, 8 ounces to be given every six hours. Twelve hours later, gave more oil and antiferments through stomach-tube. Peristalsis was good and the pain lessening. Bowel evacuations started soon afterward and the animal recovered without further treatment.

Case 20: Same horse as case 19 above. Month later, same diagnosis—impaction of the colon. Gave quart of oil and antiferments through stomach-tube, and 90 grains of barium chloride. Injected 4 cc of lentin. In this case, lentin exhibited its regular action, causing salivation, sweating, and a rapid increase in peristalsis, resulting in two bowel movements in two hours, which ended the trouble. Credit

for the recovery in this case all goes to lentin, as two hours is too soon for orally administered barium chloride to act.

Case 21: A 16-year-old mare. In very poor condition, just an old "skate." Had been treated by another veterinarian, who had given physics. She had over-eaten on rye grain. Found definite impaction of the colon. Pumped stomach out and gave oil and antiferments. Injected 3 cc of lentin and in a few minutes the animal started to breathe heavily, tremble and heave; and her eyes seemed to be "popping out." It was learned then that the animal had an asthmatic tendency, and as lentin constricts the bronchioles it is contraindicated in such cases. Subcutaneous injection of one-half grain of atropine stopped the lentin action, and in 20 minutes the animal had resumed normal breathing. Continued treatment with oil and antiferments, but the animal died six hours later.

SUMMARY

As a test of the usefulness of lentin in veterinary practice, the chemical was administered to 21 horse patients.

The action of lentin in stimulating glandular secretions was plainly evident from the induction of salivation, sweating and watery stools.

The effect of lentin in stimulating peristalsis is usually very rapid, purgation being induced frequently in 20 to 30 minutes. In severe, dry impactions purgation may be delayed several hours until the impacted mass is softened by increased secretions. In some cases a second dose, four to six hours later, is needed.

In 16 cases of digestive disturbance, mostly impactions, and in one case of azoturia in which lentin was used as a rapid-acting purgative, the results were excellent. Excellent results were also secured in one case of choke.

In two cases of severe, dry impactions, which were far advanced and apparently near to inflammation of the bowels when treatment was started, injections of lentin did not produce the usual purgation, and the two animals died. In another case of far advanced impaction, the animal revealed a "heavey" tendency after the injection of lentin, and as it is contraindicated in such cases, the action of the chemical was stopped with an injection of atropine.

CONCLUSIONS

Lentin is more effective than arecoline. It does not act more quickly, nor does it cause more pain; but it does cause freer peristalsis—evidently due to more wetting up and softening of the impacted mass by the coincidental strong stimulation of the intestinal secretions by lentin, as the stools are more watery than with arecoline. Lentin is more regular and surer in action.

Lentin is safer than other rapid-acting purgatives—arecoline, eserine, or barium chloride, because it does not depress the circulation. Lentin is safer also because its action can be con-

trolled—something that cannot be done with any other rapid-acting purgative. If desired, the parasympathetic action of lentin can be stopped instantly by a subcutaneous injection of atropine, as this drug paralyzes the myoneural junctions of the parasympathetic nerves.

Because parasympathetic stimulation constricts the bronchioles, lentin is contraindicated in heaves or in any case having an asthmatic tendency.

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POSTERIOR PARALYSIS IN SWINE*

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Posterior paralysis is a disease manifestation which is fairly frequently encountered in swine. It is now apparent that the theories generally held regarding the nature of this affection do not adequately account for all of the cases. Nutritional deficiencies already have been recognized as a cause of paraplegic symptoms in swine. Evidence has been reported indicating that a

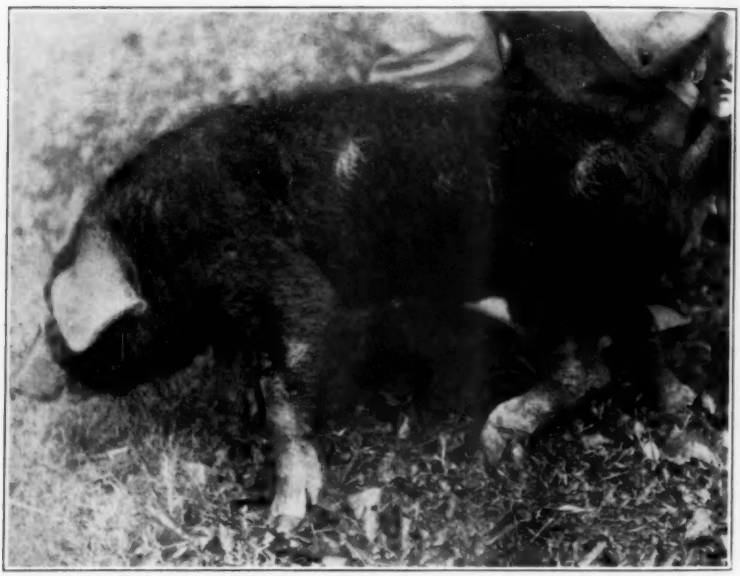

FIG. 1. A case of flaccid paralysis of the hind parts, with muscular atrophy. This type of case may result from a vertebral fracture, or from a transverse myelitis which occurs independently of skeletal defects.

deficient supply of certain vitamins may cause paralytic conditions in pigs. Moreover, it is obvious that an inadequate mineral supply, particularly in rapidly growing swine or in pregnant brood sows, may result in osteomalacia and spontaneous fracture of the vertebrae or leg bones. Fractures in the vertebral column are nearly certain to be followed by more or less paralysis. Fracture of the femur in large swine, such as brood sows, may cause symptoms which are difficult to distinguish clinically from posterior paralysis.

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Infectious Forms

The principal purpose of this paper is to call attention to evidence indicating that, in addition to the conditions which have been previously recognized, there is an infectious form of posterior paralysis in swine. This evidence has been accumulated as the result of several years of observation on the disease in the field, and microscopic studies of spinal cords and nerve trunks from affected swine.

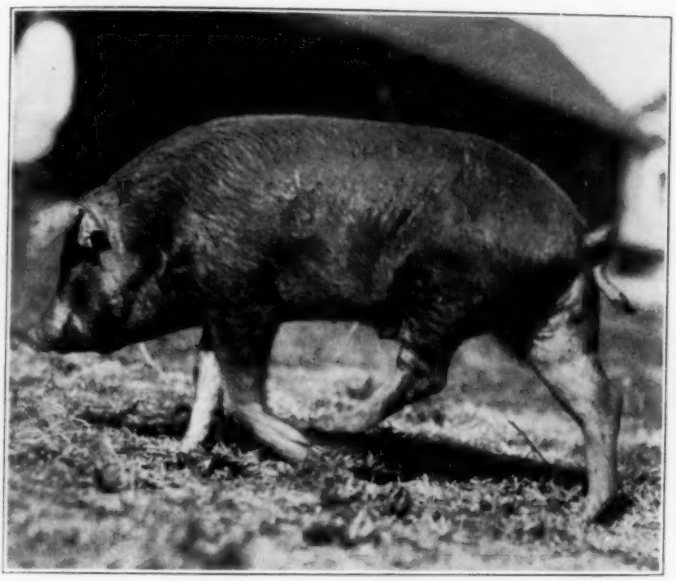

Fig. 2. A shote showing spastic or "stringhalt" symptoms. The left hind leg is raised excessively high. There was also muscular atrophy involving the gluteal and biceps muscles. These cases usually also show symptoms of more or less myelitis.

The field observations indicate that the disease is sporadic and ordinarily occurs in shotes; although it has been observed in suckling pigs. In a good many instances it is limited to pigs of one litter; and frequently affects every pig in the litter. This type of posterior paralysis has been observed to occur under a wide variety of conditions as regards feed. It has been observed in pigs which were on alfalfa pasture and were receiving, in addition, corn and tankage.

Symptoms

As an early symptom, it may be observed that the pig quickly raises and lowers, or stamps its hind feet. This premonitory

symptom is usually followed within a few days by more or less paralytic manifestations (fig. 1). The paralysis may vary in degrees from a slight weakness or incoördination of the hind parts to a complete loss of the use of the hind limbs. In some instances the paralysis is of a progressive type, and finally involves the fore limbs. A somewhat atypical form has been observed several times, in which the affected animal steps excessively high with the hind legs (fig. 2). These cases often also

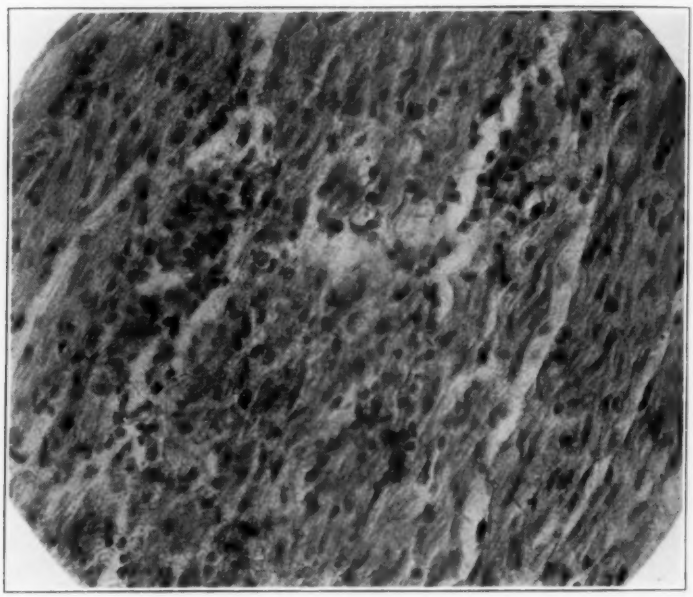

FIG. 3. Round cell infiltration of the peroneal nerve from a pig which had shown "stringhalt" symptoms. These cases often also show similar microscopic lesions in the spinal cord.

show more or less weakness or incoördination of the hind parts. A very common sequel to paralysis is an easily recognizable atrophy which may involve the biceps and gluteal muscles as well as the more distal muscles of the hind limbs.

PATHOLOGY

Microscopic studies of nerve tissues from affected animals have shown the presence of inflammatory changes which may safely be attributed to some infectious agent. It has been found that an extensive search of the spinal cord is sometimes necessary to reveal the lesion which undoubtedly accounts for the symptoms observed. Cases have been studied in which even a transverse

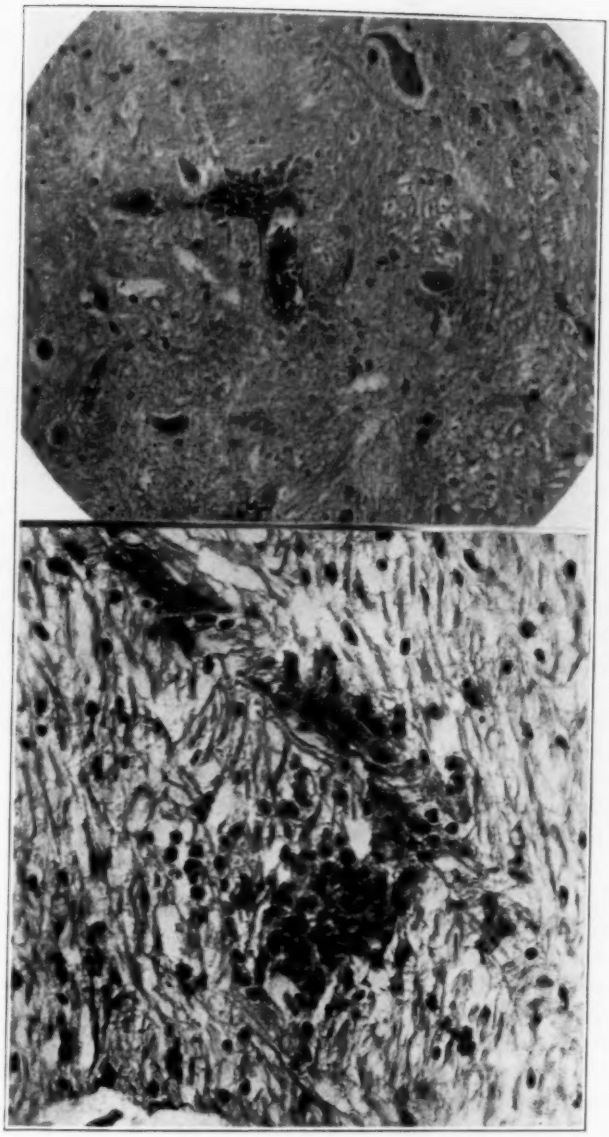

FIG. 4 (above). An accumulation of round cells in the gray matter of the spinal cord from a pig which had shown paraplegic symptoms.

FIG. 5 below). A collection of infiltrating cells in the white matter of the spinal cord from a pig which had shown posterior paralysis.

myelitis was limited to a very restricted area, as indicated by examination of longitudinal sections of the spinal cord. Examination of the large nerve trunks from the hind legs of cases characterized by spastic or "stringhalt" symptoms has shown well marked inflammatory changes, particularly in the peroneal nerves.

The microscopic change in the spinal cord is characterized by the presence of foci of round cells (fig. 3). Sometimes these cells have a perivascular location. These foci of cells have been found in both the gray (fig. 4) and the white (fig. 5) matter of the spinal cord. The same type of cell was found in the affected nerve trunks.

Diagnosis

The differential diagnosis of posterior paralysis in swine may present some difficulties. Usually a history of the feeding and management of the animals helps in arriving at a correct diagnosis. If a mineral-deficient ration has been fed, abnormal fragility of the bones and spontaneous fracture should be looked for. If a ration deficient in accessory food factors, particularly vitamins A and D, has been used, providing an adequate supply of these factors should help clear up the question as to the part played by this type of deficiency. The existence of an infectious type of posterior paralysis in swine would make it necessary to use caution in interpreting clinical observations in the field, as well as in evaluating the results of nutritional experiments. The incidence of infectious paralysis produced in swine experimentally could easily be such as to lead to erroneous conclusions regarding the causal importance of nutritional factors.

Prognosis

As regards prognosis, swine affected with posterior paralysis due to an infective type of change in the spinal cord usually do not recover completely. However, a majority of such animals can usually be brought to a marketable condition by liberal feeding of an adequate ration. As a rule, affected animals continue to show more or less weakness of the hind parts and persistent muscular atrophy. In view of the type of microscopic change found in the spinal cord, it could hardly be expected that affected animals would ordinarily make a complete recovery from this type of disease.

If an attempt is made to control an infectious type of posterior paralysis in swine, it would appear wise to make use of quarantine and sanitary measures.

A COMPOSITE DROP-REACTION SHEET FOR THE SPOT METHOD OF CONDUCTING THE WHOLE-BLOOD TEST FOR PULLORUM DISEASE*

By DONALD E. STOVER, *San Diego, Calif.*

In January, 1937, the writer[1] reported a spot method of conducting whole-blood agglutination tests with stained antigen. Promising results were obtained when this method was used in testing for Bang's disease in cattle and pullorum disease in fowls. Later work, however, has disclosed that one difficulty in interpreting test results may be encountered when blood-antigen mixtures are deposited upon single sheets of absorbent paper. This difficulty has been found to occur as a result of too slow absorption of the liquid portion of test mixtures and a subsequent tendency, in the case of positive reactions, for the smaller clumps of bacteria to spread out and become barely visible instead of remaining in one mass in the center of each spot. With a view to eliminating this flaw and thereby improving the spot method, particularly as it may be applied to testing for pullorum disease in fowls, a composite drop-reaction sheet has been devised. This sheet consists simply of a thick piece of white filter paper and a piece of lens paper placed face to face and attached at the corners.

PREPARATION OF THE SHEET

The cost of materials used in preparing composite sheets is to be seriously considered, but as the cheaper grades of filter paper used thus far have not proved very satisfactory for the test, the writer has used a comparatively expensive grade of paper.

A rectangular sheet of drop-reaction filter paper (Whatman No. 120) is given a ½-inch border and is ruled into ¾-inch squares. Over the ruled surface is placed a sheet of lens paper which is slightly smaller in width and length than the filter paper. Corners of the lens paper are now pasted to the filter paper. If the two papers are fastened together smoothly, the composite sheet appears almost as one piece of paper, and squares on the filter paper are easily visible through the lens paper.

An inexpensive absorbent paper of some kind may prove to be just as satisfactory as the drop-reaction filter paper, provided the thickness and absorbability approximate those of this paper. The grade of lens paper chosen is even of greater importance, and if the spot method were ever used generally, the standardiza-

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tion of a paper of this type would be absolutely essential. When lens paper of different grades is examined carefully, preferably under a microscope, many slight variations in texture will be noted. Any lens paper which has a comparatively close, even texture will be found to give uniformly good results. Perhaps a paper made especially for the purpose, however, would be superior to any lens paper that is now available.

The Test

The stained *Salmonella pullorum* antigen introduced by Schaffer, MacDonald, Hall and Bunyea,[2] in 1931, and now used exclusively in the conventional method of conducting the whole-blood test for pullorum disease, may be used also in the spot method of conducting the test after the incorporation of one other ingredient. A small quantity of potassium oxalate (3 cc of a 20 per cent solution of potassium oxalate to each 100 cc of antigen) is added to the antigen to serve as a blood anticoagulant.

A drop of antigen containing the anticoagulant is placed in each of as many shell vials (size 8 x 25 mm or 9 x 30 mm) as there are birds immediately to be tested. A drop of blood from each bird is then deposited upon the antigen in the proper vial. To permit thorough mixing and to prevent coagulation of the blood, each vial is rotated quickly in the fingers as soon as the blood is added. If the weather is cool, the rack containing the vials may then be placed for six or eight minutes in a small incubation chamber similar to those now in use for whole-blood testing. Upon removal from the chamber, the rack should be tipped slowly to one side several times in order that the test mixtures may flow part way along the one side of the vials. This maneuver serves to hasten agglutination in the case of positive reactions. Shaking the vials or any other motion at this time which will cause the liquid to flow over too broad a surface is liable to disrupt the test, especially if clumps of bacteria in positive reactions tend to adhere to the sides of the vials. With the aid of a straight-tipped medicine dropper, a drop of each blood-antigen mixture is now dropped upon the ruled surface of the composite sheet. The dropper must be rinsed thoroughly after the handling of each test mixture.

Accurate interpretation of results is facilitated by the formation of spots of two or three rather distinct types. In a negative reaction, the mixture spreads evenly to form a brownish, purple spot. In a strongly positive reaction, the blue-stained bacteria which have agglutinated remain in a small, compact mass in the position where the drop struck the sheet,

while the blood and the liquid portion of the antigen spread out from the central blue spot to form what essentially is a blood spot. The spot formed in the positive reaction has the appearance of a target, with the blue spot in the center resembling the bull's-eye. Slight agglutination is indicated by the formation of a light blue spot in the center of a blood spot which is tinged with purple.

If serum instead of whole blood is being tested by the spot method, the only difference to be noted in making the readings is the absence of blood color in the spots.

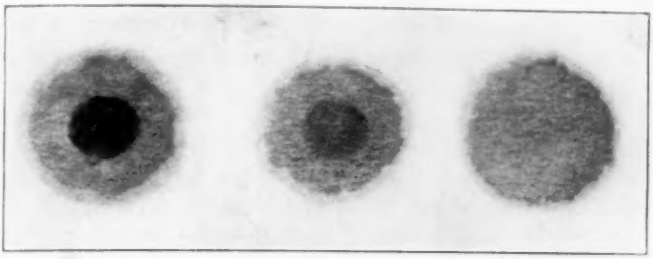

FIG. 1. Section of a composite sheet showing three different types of spots formed in conducting the agglutination test for pullorum disease. Reading left to right: a strongly positive, a slightly positive and a negative reaction.

DISCUSSION

The composite drop-reaction sheet used in the manner described is superior in several ways to the single sheet. The lens paper portion of the composite sheet serves as a filter, so that clumps of bacteria suspended in a blood-antigen mixture become enmeshed in the lens paper, while the liquid containing the blood cells and individual bacteria passes through and onto the filter paper. When the drop of liquid contacts the two papers simultaneously, it spreads very rapidly by capillarity in the air space between and is quickly absorbed by both papers. The filtration and the rapid spreading of the liquid by capillarity contribute toward the improvement of one of the more desirable features of the spot method, namely, the massing together of clumps of bacteria and their separation from the blood. Still another advantage offered by the composite sheet is the action of the lens paper in masking the spot formed by the bulk of the liquid on the filter paper beneath. In the case of a positive reaction this masking of the lower spot permits the small blue spot on the lens paper to stand out very prominently against a comparatively light background.

The automatic recording of tests probably is one of the foremost advantages of the spot method, and the use of the composite sheet, which is slightly less substantial than the single sheet, in no way serves as a barrier to making permanent records. Composite sheets bearing test results may be labeled and filed away for future reference.

The avoidance of coagulation and too rapid evaporation of the blood-antigen mixtures, the massing together of clumps of bacteria in a position away from the blood, and the automatic recording of tests all constitute very definite assets. In contrast to the conventional method, the spot method in which the composite sheet is employed would seem to go a step farther toward making the whole-blood test complete and more dependable.

SUMMARY

With a view to eliminating one outstanding flaw in the spot method of conducting the whole-blood test for pullorum disease, a composite drop-reaction sheet has been devised and put into use. This sheet consists simply of a piece of drop-reaction filter paper and a piece of lens paper placed face to face and attached at the corners with paste.

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Man o' War

The famous horse, Man o' War, reached his 20th birthday on March 29. It is reported that approximately 850,000 people, from practically every country of the globe, have visited the stable at Faraway farm near Lexington, Ky., to see this famous race horse. He has never had a sick day in his life, and is given a six-mile gallop every day to keep him in good condition. Man o' War won 20 of the 21 races he ran during the two years he was on the track, and about a quarter of a million dollars for his owner, Samuel D. Riddle. Since retiring to the stud, "Big Red" has sired 236 horses that have won more than two million dollars.

Pure friendship is something which men of an inferior intellect can never taste.—LaBruyere.

URINARY CALCULI IN FUR-BEARERS*

By EARL F. GRAVES, *Poynette, Wis.*
Wisconsin State Fur Farm

The finding of urinary calculi in the kidneys, ureters, urinary bladder or urethra is a very common occurrence in autopsies on ranch-raised fur stock. Unfortunately it is rare to get an intelligent history on the average case that is brought in. In practically every case the owner simply finds the animal dead. Only once or twice were observations made prior to death. In these cases it was reported that the animals appeared to attempt to micturate often but could not do so. The following are only a few of the cases which have been brought to the laboratory:

Case 1: Mature female. Found dead by the owner. The smaller stones shown in figure 1 were found in the urinary bladder. The large one occupied the right kidney. Total weight was 10 grams.

Case 2: Mature male raccoon. Excellent condition. No symptoms observed by keeper. Found dead in nest-box one morning. The stone shown in figure 2 occupied the entire urinary bladder. Weight, 5.2 grams. There were many minute stones which adhered to the bladder walls and have never been removed to be weighed.

Case 3: Young but matured male. Three stones (fig. 3) were found just beyond the mouth of the urinary bladder, in the urethra. Weight, 0.2 grams.

Case 4: Male, matured mink. Found dead by the owner. No history. On autopsy two large stones (fig. 4) were found, one in each kidney. Upon removal, the smaller of the two stones was the same size as the larger one but suddenly it burst in my hand and the many fragments were lost in the sand where I was standing. Careful search was made but they could not be found. The weight of the remaining piece and the other entire

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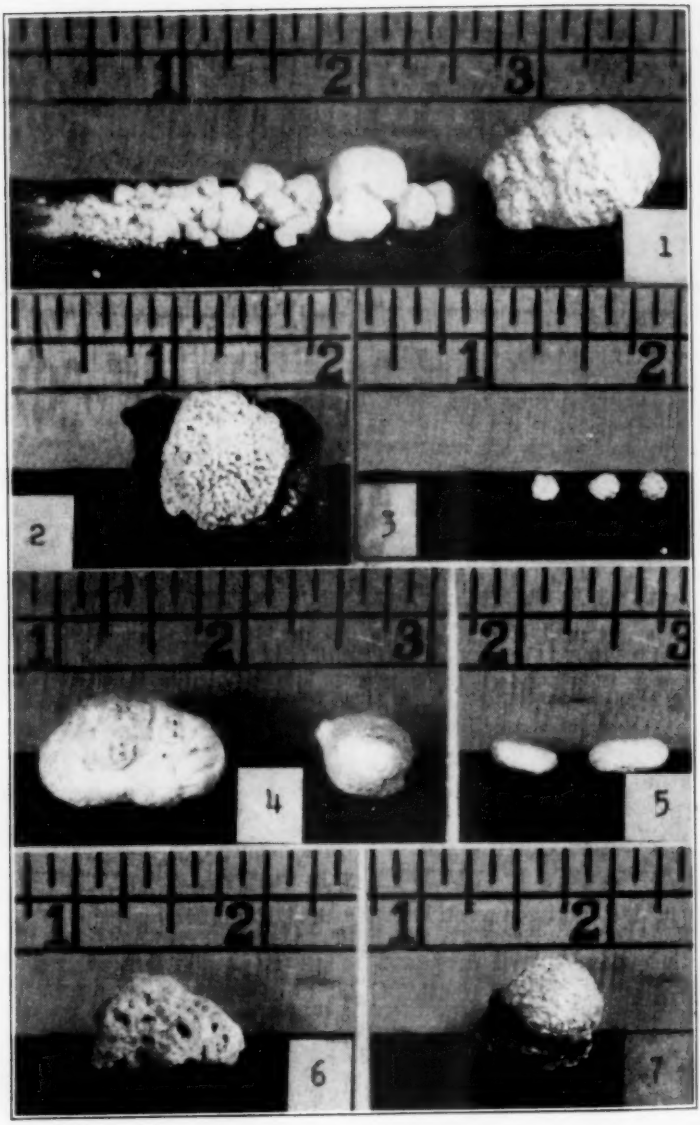

FIGS. 1-7. Urinary calculi found in fur-bearing animals.

stone is 4.9 grams. Nothing remained of the functional tissue and the stones occupied the entire capsule in each kidney.

Case 5: A matured male mink. No history. Autopsy showed two calculi in the urethra (fig. 5). Weight, 0.2 grams.

Case 6: Mature mink, sex not recorded. Large calculus (fig. 6) was found occupying all of one kidney. No functional tissue remained. Weight, 2.2 grams.

Case 7: Matured mink, sex not recorded. No history. The calculus shown in figure 7 occupied the entire urinary bladder. Weight, 1.2 grams.

In all of these cases the owners claimed that they had observed no clinical symptoms and that they found the animals dead in their pens or nests. In none of the cases was there severe emaciation but the stock was in fair to excellent condition.

A number of times I have found literally a handful of urinary calculi in the urinary bladder of silver foxes. Individual stones are very commonly found in the fox.

Cholelithiasis in Mink

Once, nearly ten years ago, while performing an autopsy on a mink, I observed a biliary calculus. Since that time I have actually posted thousands of mink and foxes but have never run across another cholelith in either a fox or mink.

PARAGONIMUS WESTERMANI IN MINK*

By Earl F. Graves, *Poynette, Wis.*
Wisconsin State Fur Farm

Subject: Male mink, mature, age not known as animal was wild caught. The animal was sent to the state farm as a confiscated mink. Upon presentation, it appeared perfectly normal but was placed in isolation. After two months, the keeper reported that the animal was off feed. It was brought into the laboratory. Posterior paralysis and profuse diarrhea were in evidence. The eyes were bright but the appetite was indifferent. An examination of fecal material showed a heavy infestation of *Paragonimus westermani.*

Treatment: Tomato juice was substituted for drinking water and a ration containing plenty of vegetables given. Within two days, the animal became so ill it appeared to be dying. After remaining comatose for a day, it seemed to become markedly

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improved, ate and drank well. At no time was there a bloody nasal or oral discharge but at times the mink did drool profusely. No symptoms of pneumonia were observed. While I had no hope of the animal effecting a recovery, I was of the opinion that there was considerable improvement in a general way when one night it suddenly died.

Postmortem: Autopsy revealed much lung congestion and several enlargements or cysts which contained a considerable number of the flukes. This is the second case of *Paragonimus westermani* in mink to be brought to me. I believe it to be a rather common affliction, for I frequently find the ovum in mink fecal material which is sent in for examination.

MILK FEVER: ITS COMPLICATIONS AND TREATMENT*

By DANIEL P. EHLERS, *Butler, Pa.*

A good sub-title for this subject would be "The Dairy Practitioner's Dilemma." This has been written for the sole purpose of stimulating interest in those who are successful in handling this condition and all of its complications, to the point of writing their experiences for the benefit of those of us who still curse roundly when one of our best clients calls up to say, "She ain't up yet, Doc."

Several years ago, we learned the advantage of the old feeble 20 per cent solution of calcium gluconate. Quite a high percentage of cases treated with this material had to be retreated. Then came the 23 per cent solution, which was hailed as the "full-dose treatment." It had some advantages, but still some cases failed to respond. Then the manufacturers made up the volume to as much as 500 cc, but still a number of patients stayed down. At about this same time, some of the pharmaceutical houses offered a 30 per cent solution, and in our hands this gave the best results. We explain it on the basis of shock to the calcium-utilizing apparatus. Still, we had those cases that were not quite "out cold," and these failed to respond in many cases to this latter solution, the same as had been the case with all previous preparations.

One hundred cc of a 50 per cent glucose solution, given after the 250 cc dose of 30 per cent calcium, seemed to be the answer to this. However, when we tried a solution that contained still

*Received for publication, March 2, 1937.

more of the calcium gluconate, and twice this dose of glucose, we had to retreat more cases than ever before. Parathyroid extract was a total failure in our hands. We tried a solution of calcium, made by one of our best houses, that was not suspended by boric acid, and we not only failed to get any response, but produced the first and only sloughed vein we have ever had. We have tried calcium gluconate tablets, calcium chloride in solution *per os* and raw ground lime and molasses on the feed.

It is our belief that our practice is a little unusual in that a large part of our work is on farms owned by individuals who are employed by the steel and oil industries in this territory. This means that they are in a financial position to indulge in cattle that are quite uniformly of a very high quality. We see calcium deficiencies in all of their complications, from the animal that is breathing her last in a complete coma, to those that eat and drink regularly, have normal peristalsis and milk-flow, but simply have difficulty in getting up. It is our opinion that these cannot be handled as a group, with one type of treatment, but must be carefully selected and treated as individual problems.

Roughly we divide our cases and treat them as follows:

Type 1: The new case, just coming on. We give this type of animal 500 cc of a solution containing 2¾ oz. of calcium gluconate and 3½ oz. of glucose. She usually responds without trouble, in about two hours.

Type 2: The comatose case. After support with ½ gr. of atropine sulfate, she receives 250 cc of 30 per cent calcium gluconate, followed by 100 cc of 50 per cent glucose. She usually wants to get up while we are cleaning the injection apparatus.

Type 3: The case that cannot get up, but is perfectly conscious. No peristalsis. May even eat a little. Usually down for two days or more, and possibly complicated with mechanical pneumonia from being drenched with various panaceas by the neighborhood handy-man. This type presents the greatest problem. We give her the 30 per cent solution and leave instructions that as soon as she gets up she shall be given a full dose of the saline laxative that we furnish. The owner, in about 50 per cent of these cases, caters to our conceit by calling the next morning to say that the cow is worse than she was before we treated her. So we return and repeat the injection, possibly passing the stomach-tube to give either a saline purge or two quarts of mineral oil, as the case seems to demand. We go home knowing that we will hear from her again. She usually lies around for several days, and then one morning gets to her feet as if nothing had happened. The bowels act as if no laxative had ever been near her. Some-

times it is necessary to follow up with daily doses of mineral oil, but this is not the rule. Are we correct in assuming that this type does not respond because of the intestinal stasis?

Type 4: The case that is apparently normal in every respect, except that she has difficulty in getting up. Usually an exceedingly heavy milker, and usually of the Holstein breed. The various solutions have no effect on these cases for more than a few hours. Her bowels may be a little "costive," as the farmers say. We get the best results by giving one ounce of raw ground lime and a tablespoonful of molasses on the feed thrice daily. In several weeks, she shows a grand improvement, but on freshening the next time, she will probably come down with a typical case of milk fever.

Type 5: The frantic, demented case. We have seen only three of this type. They are usually loose in the barn after breaking the stanchion or neckchain. They froth at the mouth, roll the eyes and stagger around, even attacking other cattle or men. The masseter muscles are usually in tonic spasm. The first one we saw we took to be intoxicated from forage, but when she did not respond to drastic purgatives, we gave her a dose of the 30 per cent solution in desperation, and had perfect results in a very few minutes. We feel that these cases are slow ones, where the bowels are so statical that auto-intoxication complicates the clinical picture.

We have learned not to expect results in any milk fever case when there is a local pyogenic process anywhere in the body, such as a streptococcic mastitis. We can return the patient to consciousness, but she will not respond completely until the infection is greatly improved.

Have we correctly classified these cases? What can be done to improve our treatments? We are sure that there are many practitioners who can discuss this matter through the JOURNAL in a way that will be beneficial to all large-animal men who must care for good dairy cattle.

HEATED MILK IN CALF SCOURS*

By W. E. MARTIN, *Wapakoneta, Ohio*

I have read with interest the article in the February issue of the JOURNAL, entitled, "The Use of Acidophilus Milk in the Treatment of Dysentery of Young Animals."

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In the community in which I have practiced for many years, there used to be many old German farmers. Their remedy for scours in calves was to drop a red hot horse shoe in the calves' milk. I modified this treatment by heating the milk almost to the boiling point. This would be the same, minus the small amount of iron which might be dissolved from the horse shoe.

While acidophilus milk is doubtless beneficial in intestinal diseases of young animals, I am wondering how much of the benefit is derived from the heated milk.

Bang's Disease Regulation Adopted by Fair Association

It is interesting to note that the International Association of Fairs recognizes the importance of not allowing the exhibition of cattle unless they are free from Bang's disease.

A resolution was adopted at the recent convention in Chicago, which specifies that all breeding and dairy cattle over six months of age exhibited at fairs, which are members of the Association, must have passed a negative test for Bang's disease within 60 days prior to the opening day of the fair, except such cattle as originate in herds designated and certified by the proper sanitary officials of the state as abortion-free herds. The tests must have been applied by veterinarians approved by the live stock sanitary authorities of the state wherein the cattle are tested.

Price Relationships

Unless directly interested in such matters, the average individual is not likely to connect such things as the market prices for cottonseed oil and pork chops. Cottonseed oil may be used in place of lard in many products. During the years 1932 to 1935, the price of lard fluctuated from less than four cents per pound to more than 17 cents. The average hog yields about 35 pounds of lard. Hogs slaughtered in the United States during 1935 yielded over 600,000,000 pounds of lard. When the price of lard gets too high, cottonseed oil is used in its place. The price of lard varies with the price of hogs. Very intimately associated with the price of hogs is the price of corn. Usually, if hogs are selling at ten dollars or more per hundredweight and corn at a dollar or less per bushel (10 to 1 ratio), it is more profitable to feed the corn to hogs, and sell it as pork, than to sell the corn directly.

THE SAINT BERNARD. Joseph H. Fleischli, President of the Saint Bernard Club of America. 1st edition. 106 pages, with 63 illustrations. Judy Publishing Co., Chicago, 1936. Cloth, $2.50.

The author is of Swiss parentage and it is not difficult to understand his fondness for and his knowledge of the Saint Bernard, a breed which had its origin in Switzerland. He describes the controversy over the standards which was brought about through the different characteristics of specimens of the breed raised in England, Switzerland and the United States.

The text contains stories of the saving of lives by these dogs during storms. He points out the peculiarities which fit this breed for life-saving. The Saint Bernard has an unfailing sense of direction, an extraordinary sense of smell, presentiment and endurance in severe weather.

In the chapter on diseases, Mr. Fleischli emphasizes the desirability of consulting a veterinarian when illness appears instead of waiting until the dog is near death. He advises the dog-owner to obtain his medicinal agents from the veterinarian.

LABORATORY METHODS OF THE UNITED STATES ARMY. Majors James S. Simmons and Cleon J. Gentzkow, Medical Corps, U. S. Army, Washington, D. C. 4th edition. xix + 1091 pages, with 70 illustrations. Lea & Febiger, Philadelphia, 1935. Limp binding, $6.50.

The fourth edition of Laboratory Methods of the United States Army was prepared at the Army Medical School by medical and veterinary officers. This edition follows the same arrangement as the previous editions. The text is designed to meet the need of the laboratory worker who must have his data in compact form and readily available for quick reference.

Some parts of the book have been revised, but most chapters have been entirely rewritten. The title might lead one to infer that only laboratory methods were included in the text, but the book contains material other than strictly technical procedures.

In the section on bacteriology, tables of classification and brief summaries of the more important characteristics of certain bacteria have been described. In the discussion of protozoölogy, helminthology and entomology, the text has been enlarged and includes illustrations of lice, fleas, mosquitoes and ticks.

A new section on Rickettsia and Filtrable Viruses and another on Statistical Methods have been added. Tests for typhus fever and Rocky Mountain spotted fever are outlined. The text presents a recent classification of filtrable viruses and outlines certain practical methods which can be used for the collection, preservation, study and identification of specimens from animals or individuals affected by virus diseases.

Procedures for examination of pathological specimens, chemical technic, toxicological methods and special veterinary laboratory methods are fully outlined for the laboratory technician.

HANDBOOK FOR VETERINARY SURGEONS. Fred Bullock, LL. D., Registrar, Royal College of Veterinary Surgeons, London. 3rd edition. 302 pages. Baillière, Tindall & Cox, London, 1936. Cloth, 7s. 6d., net.

The third edition of the Handbook for Veterinary Surgeons has been carefully revised and enlarged. The author has rearranged part of the contents of the text in order that it may be used as a guide for students in veterinary jurisprudence. It may also be adopted for the use of veterinary surgeons, in Great Britain, Ireland and Scotland, as a legal reference book.

Although the text does not apply specifically to conditions in the United States, many lessons for the good of the profession may be obtained through a close study of it. The English law tolerates unqualified practitioners (non-graduates), but will not allow them to use any title which might mislead the public. Their regulations are such that veterinarians are encouraged to become colleagues instead of competitors. The text outlines professional conduct for graduate veterinarians. If the rules are strictly observed, not only will the interests of the profession be served, but also those of the public.

Organization is urged for young graduates. They are advised to cultivate both social and scientific intercourse with other members of their profession. The belief is that the profession flourishes best when its progress is stimulated by the interest of a large proportion of the individuals who compose it.

MEDICAL SCIENCE EXHIBITS. A CENTURY OF PROGRESS. Eben J. Carey, Sc. D., M. D., Dean, Marquette University School of Medicine, and Chief, Medical Section, Hall of Science, A Century of Progress, 1931-1934. 204 pages, with 155 illustrations. A Century of Progress, Chicago, 1936. Cloth, $2.00.

In this book, Dr. Carey has presented a pictorial review of the exhibits shown in the Medical Section of the Hall of Science at A Century of Progress Exposition in Chicago, 1933 and 1934. The exhibits illustrate the tremendous strides made during the past century in the causes, detection, treatment and prevention of human and animal diseases.

Members of the American Veterinary Medical Association will recall the exhibit shown at the exposition by the Association. The general scheme was an arrangement of eight dioramas depicting the most important phases of the veterinary profession. The legends in front of the dioramas explained briefly military medicine, general practice, research, veterinary education, small-animal practice, biological manufacture, sanitary science and public health.

THE LEGAL ASPECTS OF MILK CONTROL. James A. Tobey. 1st edition. 102 pages. International Association of Milk Producers, Chicago, 1936. Cloth, $3.00.

The preparation of this text was for the purpose of presenting certain legal information to executives of the dairy industry, milk control officials, and attorneys concerned with the control of milk and milk products. The author, who is a member of the bar and a public health worker, shows how court decisions have recognized milk as a necessity and have given their decisions against violations for adulteration and concentration.

Dr. Tobey states that the tendency of the courts has been to uphold state and municipal authorities when a standard for milk and dairy products has been violated. The text outlines the authority for the inspection of dairies which are beyond the city limits, such inspection being justifiable and proper for the protection of the public health.

The same legal principles which apply to the control of bovine tuberculosis will hold good for the control of Bang's disease by states and their political subdivisions. In most decisions involving pasteurization of milk, the ordinances have been upheld.

The chapter on liability in connection with dairy products contains sections on the liability for unwholesome dairy products,

deliveries to schools and hospitals, broken glass in milk, diseases due to milk, and other subjects.

Veterinarians engaged in milk inspection will find this text a convenient reference work for information on many legal questions involved in the sanitary control of milk.

VETERINARY MEDICINE AS A CAREER. Research Booklet No. 71. 19 pages, with illustrations. The Institute for Research, Chicago, 1936. Paper, $1.00.

This brochure is designed to assist students in obtaining information concerning the veterinary profession. The subject is outlined so that young people who are seeking a vocation may compare the possibilities of veterinary medicine with other professions.

The text defines veterinary medicine, gives some of its history and the educational qualifications required for its study. Considerable space is devoted to the advantages, disadvantages, opportunities and earnings of the veterinary profession.

The publication points out the importance of the veterinarian as a guardian of public health, a preserver of animal life in preventing ravages of disease among live stock on the farm and the ranch, and as a provider of treatment for sick animals.

THE APPLICATION OF ABSORPTION SPECTRA TO THE STUDY OF VITAMINS AND HORMONES. R. A. Morton, Department of Chemistry, University of Liverpool. 70 pages, with 25 illustrations. Adam Hilger, Ltd., London, 1935. Cloth, 10s. 4d. post free.

The author has outlined studies in absorption spectra of vitamins, which will be of most value to those who are studying chemistry. The text gives descriptions of six vitamins and explains how they are affected by rays of light. The fact is admitted that spectroscopic research in this field is more empirical than in some fields of scientific study and the value of it lies in its use as an adjunct to biochemical and organic methods of study. The author states that vitamin research must always accept the animal test as the first and last courts of appeal. In the chapter on hormones, it is pointed out that little use has been made of absorption spectra in that field.

First prize for short notes to teacher goes to this one: "Please excuse Willie from school today. He caught a skunk."

The Prairie Farmer.

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The Treatment of Ascariasis in Chickens. P. P. Levine. Reprint from *Corn. Vet.*, xxvi (1936), 2, pp. 119-126.

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A Study of Some Acid-Fast Actinomycetes from Soil with Special Reference to Pathogenicity for Animals. Ruth E. Gordon and W. A. Hagan. Reprint from *Jour. Inf. Dis.*, lix (1936), pp. 200-206.

A Study of the Antigenic Properties of Certain Normal and Pathological Lymphoid Deposits in Tissues of Chickens. Carl Olson and Alexander Zeissig. Reprint from *Jour. Immunol.*, xxxi (1936), 4, pp. 309-320.

The Vaccinal Immunization of Cattle for Bang's Disease. D. B. Meyer and I. F. Huddleson. (Tech. Bul. 153. Mich. Agr. Exp. Sta., November, 1936. pp. 25.)

The Etiology of Fowl Paralysis, Leukemia and Allied Conditions in Animals. V. The Oral Exposure of Chickens Infected with Various Species of Eimeria (Coccidia) to *Salmonella aertrycke*. VI. The Oral Exposure of Chickens Infested with Ascaridia, Taenia and Capillaria to *Salmonella aertrycke*. (Tech. Bul. 305. Fla. Agr. Exp. Sta., November, 1936. pp. 66.)

A Field Condition Resembling Nutritional Encephalomalacia in Chicks. Erwin Jungherr. Reprint from *Sci.*, lxxxiv (Dec. 18, 1936), 2190, pp. 559-560.

National Research Council. Organization and Members, 1936-1937. (Washington, D. C., 1936. pp. 67.)

Tuberculosis Eradication and Live Stock Loss Prevention, 1936 Progress Report. Harry J. Boyts. (Sioux City, Iowa, 1937. pp. 18.)

The Eradication and Control of Live Stock Diseases and Prevention of Losses Incident to Marketing Live Stock, 1936 Annual Report to the Live Stock Sanitary and Loss Prevention Board of the Omaha Live Stock Exchange on. W. T. Spencer. (Omaha, Neb., 1937. pp. 21.)

Classification of *Bacterium coli* from Diseased Calves. Reginald Lovell. Reprint from *Jour. Path. & Bact.*, xliv (1937), 1, pp. 125-139.

Horse and Mule Power in American Agriculture. (Book 228. Horse and Mule Association of America, 1937. pp. 71. Illus. Price, 10c.)

The Chemistry of Stock-Poisoning Plants. James Fitton Couch. Reprint from *Jour. Chem. Ed.*, xiv (1937), 1, pp. 16-30.

Pennsylvania Bulletin, University of. School of Veterinary Medicine, Announcement, 1937-1938. (Philadelphia, Pa., 1937. pp. 64.)

THE PROBLEM OF INFECTION AS PRESENTED BY BACTERIAL INVASION OF THE CHORIO-ALLANTOIC MEMBRANE OF CHICK EMBRYOS. Ernest W. Goodpasture and Katherine Anderson. Amer. Jour. Path., xiii (1937), p. 149.

Inoculation of the chorio-allantoic membrane of chick embryos with pure cultures of pathogenic bacteria is a practical method for studying many problems of infection, especially the early stages of invasion. It is indicated in this survey of the method of studying bacterial infection in chick embryos that many pathogenic bacteria find in either mesodermal cells (fixed or mobile) or epithelial cells, or both, favorable and possibly necessary media for invasion of the living host. In these instances phagocytosis instead of representing resistance to infection actually favors it. Among these pathogenic bacteria that are able to utilize a living intracellular environment for growth are *Streptococcus veridans, Aerobacter aerogenes, Brucella abortus* and *Mycobacterium tuberculosis* avium. *Staphylococcus aureus* and *Streptococcus hemolyticus* may be in part destroyed by the phagocytes of the embryo and they appear to be incapable of growing in an intracellular medium in this host.

THE HISTOPATHOLOGY OF NATURAL AND EXPERIMENTAL CANINE DISTEMPER. W. A. DeMonbreun. Amer. Jour. Path., xiii (1937), p. 187.

The virus of canine distemper has a definite affinity for vascular endothelium and for cells of the reticulo-endothelial system. The virus spreads in the body of the host mainly by way of the blood-stream. There is no evidence that it passes along the nerve branches. The natural route in infection is by way of the respiratory tract. Nuclear inclusions, heretofore unreported, occur in liver cells, bronchial epithelial cells, glandular cells of the stomach and intestine, and bile-duct epithelial cells and cytoplasmic inclusions in bile-duct epithelial cells. Various clinical types of the disease have been induced in puppies with a single

strain of virus. Among the clinical types of distemper discussed are catarrhal, nervous or encephalitic, intestinal and tegumentary. A rapidly fulminating form, the acute encephalomyelitic type, characterized by extensive involvement of the vascular endothelium by the virus, is fairly common in young puppies but often passes unrecognized because of its short course. Interstitial pneumonia, follicular necrosis in the spleen with proliferation of reticulo-endothelial cells and nuclear inclusions in some of these cells and the same type of inclusions in reticulo-endothelial cells of the swollen mesenteric lymph-nodes characterized naturally acquired distemper in young puppies with and without symptoms referable to the central nervous system. The histopathology and cytology of the disease in dogs and ferrets are similar.

NUTRITIONAL EDEMA IN THE DOG. IV. Peptic ulcer produced by the same low protein diet that leads to hypoproteinemia and edema. A. A. Weech and B. H. Paige. Amer. Jour. Path., xiii (1937), p. 249.

The development of peptic ulcers of the stomach and duodenum in dogs maintained on a diet deficient in protein occurred in the experiments described. Among 22 animals that received the diet for an average period of 90 days there were eight (36 per cent) that exhibited true peptic ulcers at autopsy. Of the remaining 14, there were five (23 per cent) that showed erosions of gastric or duodenal epithelium without true ulcer formation.

STUDIES ON BLOOD COAGULATION. V. The coagulation of blood by proteolytic enzymes (trypsin, papain). Harry Eagle and Tzvee N. Harris. Jour. Gen. Physiol., xx (1937), p. 543.

Crude or crystalline trypsin in proper concentration causes the blood or plasma of human beings, dogs, rabbits, guinea pigs and horses to coagulate. It does not clot the fibrinogen directly but reacts with prothrombin to form thrombin. Since trypsin thus has the same effect as the physiological system, calcium plus platelets (or calcium plus tissue extracts), it is suggested as a hypothesis that the latter system contains a proteolytic enzyme with a special affinity for thrombin. The proteolytic enzyme papain also coagulates blood. Papain does not activate prothrombin but acts directly on fibrinogen to form a fibrillar gel resembling fibrin. The author suggests that thrombin, the physiological coagulant, may be a proteolytic enzyme with a specific action on fibrinogen.

BLOOD PLASMA PROTEIN REGENERATION AS INFLUENCED BY INFECTION, DIGESTIVE DISTURBANCES, THYROID AND FOOD PROTEINS. A deficiency state related to protein depletion. S. C. Madden, P. M. Winslow, J. W. Howland and G. H. Whipple. Jour. Exp. Med., lxv (1937), p. 431.

Dogs previously subjected to plasmapheresis (bleeding with the return of washed blood cells to bring about a state of low plasma protein) during long rest periods appear to increase their stores of plasma protein-building materials and their blood plasma protein concentrations above former normal levels. A sterile abscess (turpentine) induces a marked reduction in plasma protein regeneration in such dogs. Digestive disturbances seriously interfere with plasma protein production. Whereas large quantities of live-yeast upset digestion and form no plasma protein, autoclaved yeast is well utilized. Iron and liver extract (parenteral) do not influence the output of plasma protein. The proteins of red blood cells are poorly utilized for plasma protein formation. Kidney, rice polishings, a digest of beef stomach, powdered serum and powdered thyroid show a varying but not particularly distinct effect upon plasma protein production. Long periods (25 to 30 weeks) of plasma depletion remove much protein from body fluids and tissues associated with the depletion. The dog loses appetite and may vomit. There is loss of hair, skin ulceration, and a lowering of resistance to infection. The plasma protein output may fall to fasting levels in spite of food intake sufficient to maintain weight. This is considered a deficiency state related to severe depletion of the essential protein matrix of the body cells.

LIVER FUNCTION AND BLOOD PLASMA PROTEIN FORMATION. R. E. Knutti, C. C. Erickson, S. C. Madden, P. E. Rekers and G. H. Whipple. Jour. Exp. Med., lxv (1937), p. 455.

Normal dogs and two Eck-fistula dogs receiving a daily diet containing an average of one gram of vegetable protein per kilo of body weight showed, after average intervals of seven to nine weeks, slight decreases in amounts of circulating plasma protein. A third Eck-fistula dog under similar circumstances was unable to maintain its plasma protein concentration above the edema level. The animal showed active thirst and diuresis as compared with controls; biopsy showed an abnormal liver while other organs appeared normal. This dog had less than one-tenth the capacity of the normal dog to form new plasma protein when

various food proteins were added to the basal diet and no significant quantitative differences in the relative potency of these foods (liver, kidney, heart muscle, soy bean, salmon) could be distinguished. The liver abnormality was considered responsible for this abnormal reaction. This observation gives strong support to the opinion that the liver is actively concerned with fabrication of new plasma protein.

THE DISTRIBUTION IN THE BLOOD AND LYMPH OF PNEUMOCOCCUS TYPE III INJECTED INTRAVENOUSLY IN RABBITS AND THE EFFECTS OF TREATMENT WITH SPECIFIC ANTISERUM ON THE INFECTION OF THE LYMPH. Madeleine E. Field, Morris F. Shaffer, John F. Enders and Cecil K. Drinker. Jour. Exp. Med., lxv (1937), p. 469.

When rabbits are infected intravenously with virulent type III pneumococci, these organisms are found not only in the thoracic duct lymph but also in lymph from the cervical and leg lymphatics. The non-motile bacteria must have crossed both vascular and lymphatic endothelium in reaching the lymph. Intracellular transportation of phagocytes is apparently not the means by which this is effected. The intravenous and intraperitoneal injection of large amounts of homologous type specific antibody fails even after many hours to terminate or permanently reduce the pneumococcal infection of the lymph.

A RELATIONSHIP IN EQUINES BETWEEN AGE OF HOST AND NUMBER OF STRONGYLID PARASITES. A. O. Foster. Amer. Jour. Hyg., xxv (1937), p. 66.

Qualitative studies of the worm burdens of 86 equines, varying in age from nine to 30 years, demonstrated that the animals from nine to 15 years old were more than twice as heavily infested on the average, with strongylid parasites than were those over 15 years. From 59 animals, 15 years and under, an average of 1,219 worms per animal were recovered as compared to an average of 502 worms from 29 animals over 15 years. The authors interpret this as an age resistance of equines against strongylid parasites. This was demonstrated quantitatively for the commoner individual species. In the instance of *Strongylus vulgaris*, however, the data afforded no evidence of increased protection in the older animals. The data show there was no evidence of an age resistance of equines against stomach worms.

ANTIDIURETIC PITUITARY SUBSTANCE IN BLOOD WITH SPECIAL REFERENCE TO THE TOXEMIA OF PREGNANCY. K. I. Melville. Jour. Exp. Med., lxv (1937), p. 415.

A method for the quantitative extraction of posterior antidiuretic substance from blood with which it has been mixed *in vitro* and *in vivo* is presented. It has been found that a similarly extractable active substance may be detected as a normal constituent of dog and human blood. The data obtained from the blood of normal pregnancies and several cases of early toxemia do not indicate any causal relationship between the presence of this substance in the circulating blood and the early symptoms (hypertension, edema, albuminuria) of the toxemia of pregnancy.

BLOOD AND TISSUE STUDIES IN EXPERIMENTAL ASCARIASIS. H. E. Biester and Donald F. Eveleth. Amer. Jour. Hyg., xxv (1937), p. 135.

The general trend of the chemical constituents of the blood of pigs affected with experimental ascariasis, as shown in the average values, indicates: (1) a rise in the inorganic phosphorus, (2) a rise followed by a fall in blood sugar, (3) a fall of urea nitrogen with a simultaneous rise in amino-acid nitrogen. While swine livers as a rule contain large amounts of connective tissue, this in part at least is due to migrating parasites. Swine raised under controlled sanitary conditions fail to show well developed interlobular connective tissue. Considerable damage caused by migrating parasites to hepatic tissue follows experimental ascariasis infection. The reaction of hepatic tissue is more marked three days after infection and began to subside by the sixth day. The reaction is characterized by markedly granular and hydropic tissue and hemorrhagic foci. In the vicinity of such lesions which usually did not occur in the peripheral parts of the lobules there were dense infiltrations of myelocytic eosinophiles.

THE WHITE BLOOD CELL PICTURE IN HOOKWORM DISEASE OF DOGS. J. W. Landsberg and A. O. Foster. Amer. Jour. Hyg., xxv (1937), p. 141.

A marked leukocytosis did not follow every infection, whether cutaneously or orally administered. The route of infection did not seem to influence the degree of leukocytosis. An increase in the total white cell counts when it occurred was due apparently to an increase in the mature neutrophilic cells, the polymorphonuclear neutrophils. An increase in the eosinophiles did not occur after

every infection but when it did occur the maximum increases came at varying intervals after infection. There was little difference in the magnitude of the eosinophilic responses following oral and cutaneous administration of larvae. Lymphocytopenia occurred in some cases but was not constant and there was no appreciable change in the basophils or monocytes.

STUDIES ON MAGNESIUM DEFICIENCY IN ANIMALS. VIII. The effects of magnesium deprivation on the total and ultrafiltrable calcium and magnesium of the serum. Sibley W. Hoobler, H. D. Kruse and E. V. McCollum. Amer. Jour. Hyg., xxv (1937), p. 86.

As a result of magnesium deficiency, animals showed a pronounced drop in serum magnesium with a corresponding fall in the absolute concentration of the ultrafiltrable portion so that the percentage of diffusible magnesium was virtually unchanged. There was a moderate lowering of the total calcium level with a disproportionately lesser decline in absolute values of ultrafiltrable calcium so that the percentage of the latter fraction was slightly increased. In the periods marked by convulsions, with values for diffusible calcium in or near what may be conjectured as the critical range of magnesium tetany, the figures for diffusible calcium were neither correspondingly or invariably at their lowest nor at a point indicative of calcium tetany. Thus the infrequency with which the diffusible calcium even approached a critical tetanic range and the lack of concurrence between the lowest values for each animal and the onset of convulsions indicated that calcium-ion concentration of the blood was not connected with magnesium tetany. The tetanic syndrome of magnesium deficiency was closely associated with the extremely low concentration of magnesium ions in the blood.

What Price Cow?

"I hear you have a fine cow," said the stranger. "What will you take for her?"

"Wait a minute," replied the farmer cautiously, "are you the new assessor or has my cow been killed on the railroad?"

Detroit News.

Whatever is worth doing at all is worth doing well.—Earl of Chesterfield.

Regular Army

1st Lieut. Daniel S. Stevenson is directed to proceed to Fort Slocum, N. Y., for temporary duty until such time as will permit him to sail on transport scheduled to leave New York on or about May 19, 1937, for the Philippine Department.

Captain Ralph W. Mohri is relieved from assignment and duty at Fort Riley, Kan., effective on or about August 1, 1937, is then assigned to station at Army Medical Center, Washington, D. C., will proceed to that station and report to the commanding general for duty.

Captain Elmer W. Young is relieved from assignment and duty at the Front Royal Quartermaster Depot, Front Royal, Va., effective June 30, 1937, and directed to proceed to Fort Riley, Kan., for duty.

Major Francois H. K. Reynolds is relieved from his present assignment and duty at the Army Medical Center, Washington, D. C., effective in time to proceed to New York, N. Y., and sail on the transport scheduled to leave that port on or about July 8, 1937, for the Panama Canal Department, for duty with the Veterinary Corps.

Major Harry L. Watson is relieved from duty at Fort Sill, Okla., effective at such time as will enable him to proceed to San Francisco, Calif., and sail on transport scheduled to leave that port on or about June 12, 1937, for the Hawaiian Department, for duty with the Veterinary Corps.

The promotion of each of the following-named officers in the Veterinary Corps, to the grade of lieutenant colonel, with rank from March 24, 1937, is announced:

Major Allen C. Wight
Major Elwood L. Nye

Veterinary Reserve Corps

NEW ACCEPTANCES

Barry, James Richard.....1st Lt...215 Locust St., Waterloo, Iowa.
Geick, Harold Leo.....1st Lt...2980 S. Logan Ave., Milwaukee, Wis.
Meyer, Frank Leroy.....1st Lt...542 Evergreen, East Lansing, Mich.
Meyers, Luther D.....1st Lt...Care of Dr. A. G. Boyd, Dept. of Agr., Sacramento, Calif.
Riddell, Wm. Kenneth.....1st Lt...1909 State St., Santa Barbara, Calif.
Sutton, Karl LeRoy.....1st Lt...Morenci, Mich.

PROMOTIONS

To

Brandon, Glen Cecil.....1st Lt...103 E. Maynard Ave., Columbus, Ohio.
Gaydosh, Louis Wesley.....1st Lt...505 W. 31st St., Kansas City, Mo.
Jones, William Laurie.....1st Lt...325 Columbia, Leavenworth, Kan.
Mouw, John Englen B.....1st Lt...Edgerton, Minn.

SEPARATIONS

Moore, Eugene James.....1st Lt...Winston-Salem, N. C. Died March 10, 1937.

NEW ASSIGNMENTS TO ACTIVE DUTY WITH CCC

Jacobs, Marvin Henry.....1st Lt...Fort Bragg, N. C.
McNay, Garrett H.....1st Lt...Fort Moultrie, S. C.
Phifer, Wm. Gough.....1st Lt...Camp Beauregard, La.
Thompson, Thomas M.....1st Lt...Pres. of San Francisco, Calif.

TERMINATION OF ASSIGNMENT TO ACTIVE DUTY

Spring, Jacob Emmil.....1st Lt...Fort Moultrie, S. C.

United States Civil Service Examination

The United States Civil Service Commission announces the following open competitive examination:

ENDOCRINOLOGIST

Applications for this position must be on file with the Commission at Washington, D. C., not later than May 17, 1937.

At present, there is a vacancy in the Bureau of Dairy Industry, U. S. Department of Agriculture, with headquarters at Beltsville, Md. The entrance salary is $3,800 a year, subject to a deduction of 3½ per cent toward a retirement annuity. The duties are to direct research of the functions and secretions of the endocrine glands, including investigations in the physiology of reproduction and milk secretion of dairy cows and to coöperate with scientists of the Bureau of Animal Industry in research on the hormones connected with reproduction.

Applicants must have successfully completed a full four-year course leading to a bachelor's degree in a college or university of recognized standing. They must have had, subsequent to the completion of the required college course, five years of research in endocrinology, including the physiology of the pituitary gland.

Full information and application blanks may be obtained from the United States Civil Service Commission, Washington, D. C., from the secretary of the U. S. Civil Service Examiners, at any first-class post office, or from any United States Civil Service district office.

"Do you believe in heredity?"
"Absolutely! That's how I got all my money."

Indiana Farmers Guide.

MICHIGAN STATE COLLEGE SHORT COURSE FOR VETERINARIANS

The fourteenth annual postgraduate short course for veterinarians was held at the Michigan State College, East Lansing, January 25-28, 1937. The program was featured by laboratory demonstrations, lectures and discussions of brucellosis and pullorum disease.

The first day was devoted to demonstrations of laboratory methods of preparing rapid antigen for Bang's disease, by Drs. I. F. Huddleson and W. T. S. Thorp, and rapid antigen for pullorum disease, by Dr. H. J. Stafseth. The evening program was devoted to a discussion of problems in the use of the rapid agglutination test.

The program of the second day consisted of the following subjects:

"Studies on Viruses" (illustrated), by Dr. C. A. Brandly, Illinois Department of Agriculture, Urbana, Ill.

"Problems Peculiar to Virus Diseases," by Dr. Adolph Eichhorn, Pearl River, N. Y.

"Results of Comparative Pullorum Disease Testing in Illinois," by Dr. C. A. Brandly.

"The Importance of Uniform Standards for Biological Products," by Dr. Adolph Eichhorn.

"Practical Methods of Pullorum Disease Control," by Dr. E. W. Roberts, Ohio Department of Agriculture, Reynoldsburg, Ohio.

In the evening, a banquet to visiting veterinarians was served under the auspices of the Michigan State College Student Chapter of the A. V. M. A.

The program of the third day was as follows:

"The Rapid Agglutination Test in the Diagnosis of Brucella Infections," by Drs. I. F. Huddleson and W. T. S. Thorp.

"Present Status of Bang's Disease Control Work" and "Should the Michigan Practitioner Be Permitted to Make Official Plate Tests?" by Dr. B. J. Killham, Extension Specialist, Michigan State College, East Lansing.

"The Wisconsin Procedure for Manufacture and Approval of Brucella Antigen," "Bang's Disease Progress in Wisconsin" and "Wisconsin's Attitude Toward Brucella Vaccines," by Dr. Walter Wisnicky, Director of Live Stock Sanitation, Madison, Wis.

"Experiences and Observations with the Rapid Agglutination Test in the Control of Bang's Disease," by Dr. William A. Rader, U. S. B. A. I., Lansing.

"Vaccinal Immunization of Cattle Against Bang's Disease," by Dr. D. B. Meyer, Michigan Agricultural Experiment Station, East Lansing.

The program of the fourth day was as follows:

"Parasitology, a Subject of Importance to Veterinarians" and "Parasites of Sheep and Cattle, with Special Reference to Their Life Histories as a Basis for Control Measures," by Dr. Gerard Dikmans, U. S. B. A. I., Washington, D. C.

"Days Ahead for the A. V. M. A." and "Thirty Years of Army Veterinary Service from a Personal Standpoint," by Col. Robert J. Foster, president of the American Veterinary Medical Association, Washington, D. C.

Col. Foster's addresses were very inspiring to his hearers, as many of them had no idea how carefully plans had been prepared for including the services of veterinarians in connection with food, meat and milk inspection. His plea for financial help for veterinarians in the flooded areas of the Ohio and Mississippi valleys received immediate action. Over $200 was contributed to this worthy cause.

Dr. Ward Giltner, dean of the Division of Veterinary Medicine, Michigan State College, and his co-workers deserve much praise for their untiring efforts in preparing this program for the benefit of the veterinary profession. The attendance was greater than in former years because of an increased interest in Bang's disease. Up to this time, the practicing veterinarians in Michigan have taken very little part in the federal-state program of controlling Bang's disease.

E. C. W. S.

VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The January meeting of the Association was held at the Hotel New Yorker on Wednesday evening, January 27, 1937.

President MacKellar introduced our guest and speaker, Dr. W. S. Gochenour, of the Pathological Division, U. S. Bureau of Animal Industry, Washington, D. C., who gave us a very interesting talk on "Equine Infectious Anemia." Dr. Gochenour presented his subject in a very able manner, first giving us a very complete paper and then following it with some very interesting moving pictures which depicted various stages and phases of the disease. He emphasized the fact that swamp fever is found in every country of the world, is particularly widespread in the United States, and is confined mainly to horses and mules, but occasionally occurs in man. Dr. Gochenour pointed out that from

clinical symptoms alone, it is almost impossible to diagnose the disease. He stated that horse inoculation is the only way to be sure of making a positive diagnosis, but this, however, is impractical. Therefore, the development of a practical way to diagnose this disease accurately would be a step forward. Until this is done and some form of immunization is developed for susceptible animals, this disease will continue to be one of our most troublesome in horses and mules.

Dr. Gochenour's comprehensive paper and valuable time, so cordially tendered us, were greatly appreciated by the members of the Association.

JOSEPH B. ENGLE, *Secretary.*

CONNECTICUT VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Connecticut Veterinary Medical Association was held at the Hotel Bond, Hartford, February 3, 1937.

The Association was honored with the presence of Dr. Adolph Eichhorn, of Pearl River, N. Y., who gave a very interesting talk on "Recent Developments and Investigations of Virus Diseases." This proved to be a very instructive talk and was highly enjoyed by the members and visitors present. Dr. Harry W. Jakeman, of Boston, also discussed Dr. Eichhorn's subject.

A committee of four members, consisting of Drs. Edwin Laitinen, Chairman, Richard T. Gilyard, J. Douglas Winn and Geo. E. Corwin, was appointed by the president to be known as the Committee on Public Relations, which will coöperate with the Committee on Public Relations of the American Veterinary Medical Association.

The following officers were elected for the ensuing year: President, Dr. E. R. Dimock, Merrow; first vice-president, Dr. Edwin Laitinen, West Hartford; second vice-president, Dr. F. F. Bushnell, Manchester, and secretary-treasurer, Dr. Geo. E. Corwin (reëlected), Hartford. The Board of Censors was chosen as follows: Chairman, Dr. G. Leroy Cheney, Woodbridge; Dr. I. R. Vail, Bristol; Dr. N. W. Pieper, Middletown; Dr. R. V. Westerberg, New Britain, and Dr. E. M. Bitgood, Middletown.

Dr. Geo. E. Corwin, of Hartford, was elected delegate, and Dr. I. R. Vail, of Bristol, alternate, respectively, to the A. V. M. A. House of Representatives.

Dr. J. Douglas Winn, of Merrow, was admitted to membership.

The subject of relief to be rendered veterinarians in the flood-stricken area was discussed and by vote of the Association the sum of $50.00 was appropriated for the purpose.

GEO. E. CORWIN, *Secretary-Treasurer.*

HOUSTON VETERINARY ASSOCIATION

The regular monthly meeting of the Houston Veterinary Association was held at Dr. Frank Hecker's hospital, Houston, Texas, Thursday evening, February 4, 1937, with almost a full membership in attendance.

The program feature was a paper prepared by Dr. Guy A. Roberts, of Ciudad Trujillo, Dominican Republic, and read by Dr. J. G. Horning, of Houston, relative to tropical diseases, case reports, experiences and observations of Doctor Roberts while in the Republic as well as in Brazil. The Association extended Dr. Roberts a vote of thanks as well as an honorary membership.

DON B. STRICKLER, *Secretary-Treasurer.*

ALABAMA POLYTECHNIC INSTITUTE SHORT COURSE FOR VETERINARIANS

Sponsored jointly by the Alabama Veterinary Medical Association and the School of Veterinary Medicine, Alabama Polytechnic Institute, the thirteenth Annual Short Course for Graduate Veterinarians was held at Auburn, February 8-13, 1937, under the direction of Dr. I. S. McAdory, Acting Dean.

Records of all previous similar meetings were broken by an attendance of 150 veterinarians from eleven states. Various sessions featured municipal food inspection and grading of dairy and meat products, poultry diseases, Bang's disease, Johne's disease, bovine mastitis, control of hog cholera, parasites of swine, tuberculosis eradication and various other vital subjects.

The clinics were especially interesting, being handled by the clinic staff of the Alabama Polytechnic Institute and Dr. R. L. Anderes, of Kansas City, Mo. Various activities of the federal government were discussed and demonstrated by Drs. H. M. O'Rear, of Washington, D. C., R. E. Jackson, of Montgomery, Ala., and others.

The Army Veterinary Corps was well represented. Col. Robert J. Foster, Chief of the Veterinary Service, and president of the American Veterinary Medical Association, attended the sessions and was the guest of honor at a banquet tendered him on the night of February 11.

At the annual meeting of the Alabama Veterinary Medical Association held February 12, the following officers were chosen for the coming year: President, Dr. L. E. Beckham (reëlected), Tuscaloosa; vice-president, Dr. J. S. Andrade, Huntsville, and secretary-treasurer, Dr. I. S. McAdory (reëlected), Auburn.

A resolution was adopted by the Association, embodying a number of suggestions designed to increase interest in the A. V. M. A. as well as to enlarge its activities and usefulness. The resolution recommended: the publication of the proceedings of each annual meeting in one volume, at a cost not to exceed two dollars per member, the cost to be paid by an increase in the annual dues; that each annual convention be made self-supporting by increasing the present registration fee of one dollar; that each annual convention be managed by the A. V. M. A., and that a committee be authorized to examine facilities offered by cities asking for A. V. M. A. conventions and to make recommendations in connection therewith.

Wives accompanying the visiting veterinarians were entertained under the direction of Mrs. I. S. McAdory. The program consisted of a series of demonstrations arranged by Miss Dana Gatchess, Professor of Home Economics, Alabama Polytechnic Institute; a theatre party and visits to the Tuskegee Institute and the Fairfax and Lanett cotton mills.

I. S. MCADORY, *Secretary-Treasurer.*

LOUISIANA VETERINARY MEDICAL ASSOCIATION

The sixth annual short course in connection with the Louisiana Veterinary Medical Association was held February 9-10, 1937, at Louisiana State University, Baton Rouge. Dr. W. T. Oglesby, who is senior veterinarian at the University, as chairman of the Program Committee, did a nice job in selecting the program for this meeting.

Dr. L. A. Merillat, of Chicago, Ill., gave an interesting talk on parasites of horses and mules, with special reference to the bot. Much enthusiasm over the use of mass capsules of carbon bisulfide for combating these parasites was manifest; numerous questions were asked of Dr. Merillat. Dr. C. R. Donham, of Ohio State University, gave an unusually interesting talk on cattle practice, with particular reference to Bang's disease.

Dr. Richard A. Self, of Dallas, Texas, a leading practicing veterinarian of the Southwest, was the man of the hour in the eyes of the small-animal practitioners. He handled his subjects

well, with special attention given to fractures and the splinting of same.

Our other distinguished visitor was Col. Robert J. Foster, president of the A. V. M. A., Surgeon General's Office, Washington, D. C. His subject was "Days Ahead for the A. V. M. A." He stressed the importance of organization and of every veterinarian being a member of his state and national organizations. Our State Association is very grateful to our national organization for sending their President to visit with us.

This meeting was acclaimed by all who were present as the best yet held since the short courses were started six years ago. Drs. J. F. DeVine, Goshen, N. Y.; R. W. Williams, Eldorado, Ark.; D. M. Williams, McComb, Miss., and H. R. Wesson, of Scranton, Iowa, were other out-of-state visitors. A banquet was held at the Heidelburg Hotel the evening of February 9, attended by 75.

The election of officers for the ensuing year resulted as follows: President, Dr. Frank Collins, Monroe; vice-president, Dr. P. J. Orchard, and secretary-treasurer, Dr. C. M. Heflin (reëlected), both of Baton Rouge.

C. M. HEFLIN, *Secretary.*

HUDSON VALLEY VETERINARY MEDICAL SOCIETY

The regular quarterly meeting of the Hudson Valley Veterinary Medical Society was held at the Nelson House, Poughkeepsie, N. Y., on February 10, 1937 with 40 veterinarians present. Major E. M. Curley, President, called the meeting to order after luncheon. Among the guests was Dr. H. C. Stephenson, New York State Veterinary College at Cornell University, Ithaca, N. Y.

A business session followed. The proposed affiliation of the state and local veterinary societies was endorsed. One new member, Dr. D. E. Armstrong, of Hoosick Falls, N. Y., was elected.

Major Curley then introduced Dr. C. P. Zepp, of New York City, president of the New York State Veterinary Medical Society, who addressed the meeting on the subject, "Factors to Consider in Small-Animal Practice and Hospitalization." The speaker emphasized the importance of business management and ethical practices in conducting a hospital for small animals. He believes that attention to all details is important and necessary in order to satisfy animal-owners, as well as to be successful.

He recommended that careful case records be kept and that clients be advised of the necessity of carefully following feeding or other instructions. Much discussion followed.

Major Curley next introduced Dr. C. J. Hoyt, of Walden, N. Y., whose topic was "Dairy Practice." Dr. Hoyt referred to the danger of disease being carried from herd to herd through carelessness. He stressed the necessity of great care and cleanliness on the part of practitioners when going from farm to farm. He emphasized the advantages of informing the owner what the treatment was intended to do and thereby interesting the owner more directly. The speaker discussed the principles that he had applied in his own herd in connection with special grade A raw milk. There was some discussion concerning the use of vaccines, especially in the treatment of Bang's disease. Several case reports by various members followed.

J. G. WILLS, *Secretary-Treasurer.*

EAST CENTRAL (IOWA) VETERINARY ASSOCIATION

A dinner-meeting of the East Central Veterinary Association, held at the Hotel Jefferson, Iowa City, Iowa, February 11, 1937, was attended by 42 practitioners from 14 counties. Dr. Samuel S. Paul, of Clarence, presided.

Dr. H. E. Biester, of Iowa State College, Ames, gave a very interesting illustrated paper on "Moldy Food Poisoning in Horses and Equine Encephalomyelitis." The presentation inspired a lively discussion. Dr. Biester gave the scientific reasons for the great variability in the symptoms, lesions, course, fatality and spread of these diseases. He stated that when a certain part of the brain is affected, by either mold, enzyme, virus or toxin, identical symptoms are produced. The virus disease, chiefly of the less virulent western type, caused fewer deaths in Iowa horses during the year 1936, according to Dr. Biester. He stated that climatic conditions not favorable to the propagation of numbers of biting insects may have been the reason. Dr. Biester outlined experiments now being conducted on the feeding of corn affected with blue, green and gray molds.

Dr. C. L. Crider, of Elkader, presented a talk on "Cattle Practice," which brought out an informative and interesting discussion. His long experience, both as a general practitioner and in building up his own high-producing, pure-bred Holstein dairy herd, enabled him to present his subject from the standpoint of the herdsman as well as of the practitioner. He urged more sympathy and understanding on the part of the practitioner with

the herdsman's problems and ambitions. Dr. Crider believes that the practitioner should always strive to educate clients along certain lines, and thus improve his own knowledge by so doing. He states that lamenesses of dairy cows may be the cause of the loss of more butter fat in a dairy herd than any other condition. Hardware, rocks, infections, cuts, puncture wounds, and breaks of the horny covering are common in his practice.

Dr. James C. Carey, president of the Iowa Veterinary Medical Association, West Liberty, addressed the meeting on affairs and policies affecting the State Association. Dr. J. A. Barger, Des Moines, chairman of the Committee on Revision of the Constitution and By-laws, Iowa Veterinary Medical Association, outlined the changes recommended by the committee. Dr. C. E. Hunt, Mount Pleasant, president of the Eastern Iowa Veterinary Association, Inc., made a brief talk on organization. Dr. H. A. Seidell, State Veterinarian, Des Moines, gave a short talk on regulations of the Department of Agriculture which govern the operation of rendering plants and their trucks.

Dr. C. H. Banks, Tipton, chairman of the Committee on Equine Periodic Ophthalmia, Eastern Iowa Veterinary Association, made a brief report, as did Dr. John B. Bryant, Mount Vernon, chairman of a like committee from the East Central Veterinary Association.

Dr. J. I. Gibson, former State Veterinarian, Des Moines, made a brief talk regarding advances made in veterinary services in the 50 years he has been a veterinarian. Mr. Lee Cochran, of Iowa State University, Iowa City, entertained the members with motion pictures taken in Casa Verde National Park, Colorado.

VETERINARY ASSOCIATION OF MANITOBA

The forty-seventh annual meeting of the Veterinary Association of Manitoba was held at the Royal Alexandra Hotel, Winnipeg, February 12, 1937. There was a good attendance of members from all parts of the province as well as many officials from the provincial and federal Departments of Agriculture.

Dr. J. A. Martin, of Sperling, president of the Association, in his opening address during the morning session, dealt with the outstanding events in the affairs of the Association during the past year. The report submitted by the secretary-treasurer, Dr. Wm. Hilton, showed the financial position of the organization to be satisfactory. During the meeting, Dr. R. Lawson, of Shoal Lake, and Dr. J. G. Cruikshank, of Selkirk, who have been

members of the Association for 30 years, were elected honorary life members.

The afternoon session was opened with an address by the Hon. D. L. Campbell, Minister of Agriculture and Immigration of Manitoba. Mr. Campbell proved to be a most interesting speaker and extended to the Association the coöperation of his department in dealing with disease problems of live stock in the province. In the absence of Dr. S. M. Thompson, of Virden, his paper, "Hypersensitiveness—Its Various Phases," was presented by Dr. R. H. Lay, of Winnipeg.

The following addresses were delivered during the afternoon:

"Meat Inspection in Canada as I See It," by Dr. J. G. MacDonald, of the Federal Meat Inspection Division, Winnipeg.

"Some Observations—Poliomyelitis Epidemic of 1936," by Dr. F. W. Jackson, Deputy Minister of Health and Public Welfare of Manitoba.

"The Work Undertaken by the Institute of Parasitology, Montreal," by Dr. A. Savage, Dean of Agriculture, University of Manitoba.

"The Future of Veterinary Medicine in Manitoba," by Dr. M. T. Lewis, Saint James.

"Disease Control and Eradication in Manitoba," by Dr. R. H. Lay, District Veterinary Inspector, Federal Contagious Diseases Division.

"Trichinosis and Undulant Fever," by Dr. W. A. Shoults, Department of Health and Public Welfare of Manitoba.

The election of officers for 1937 resulted as follows: President, Dr. J. W. Fasken, Portage la Prairie; vice-president, Dr. T. G. Sprague, and secretary-treasurer and registrar, Dr. Wm. Hilton, both of Winnipeg. Members of the Council: Dr. J. A. Martin, Sperling; Dr. H. R. McEwen, Stonewall; Dr. J. W. Fasken, Portage la Prairie, and Dr. A. Savage, Dr. T. G. Sprague, Dr. Wm. Hilton and Dr. J. K. Morrow, all of Winnipeg. Auditors: Dr. E. J. Rigby and Dr. A. M. McFarlane, both of Winnipeg.

WM. HILTON, *Secretary-Treasurer.*

VIRGINIA CONFERENCE OF GRADUATE VETERINARIANS

The fourth annual conference of veterinarians, offered by the Department of Biology of the Virginia Polytechnic Institute, was held at Blacksburg, February 16-18, 1937. Forty veterinarians were in attendance. The following program was presented:

Address of welcome, by H. L. Price, Dean of Agriculture.

"The American Veterinary Medical Association," by Col. Robert J. Foster, president of the A. V. M. A.

"Gastro-Intestinal Infections of the Dog and Their Differentiation," by Dr. Norman J. Pyle, Pearl River, N. Y.

"The Endocrine System of Dogs," by Dr. C. J. Lange, Virginia Polytechnic Institute.

"Diagnosis of Mastitis" (lecture and demonstration), by Dr. L. E. Starr, Virginia Polytechnic Institute.

"Conservation of Our Natural Resources," by C. F. DeLeBarre, president of the Virginia Wildlife Federation.

"The Bot Control Program In Virginia" (lecture and demonstration), by Dr. Colby Graves, Culpeper. Discussion by Drs. L. K. Bailey, of Lexington, and J. E. Greer, of Pulaski.

"Diagnosis of Poultry Diseases" (lecture and clinic), by Drs. E. P. Johnson and Donald G. Lee, Virginia Polytechnic Institute.

"The Veterinary Corps of the U. S. Army," by Col. Robert J. Foster.

"Nutrition and Diet for the Dog and Cat," by Dr. W. J. Lentz, University of Pennsylvania, Philadelphia, Pa.

"Progress in Bang's Disease Control," by Dr. H. C. Givens, State Veterinarian, Richmond.

"Differential Diagnosis and Diseases of Small Animals" (lecture and clinic), by Dr. W. J. Lentz.

Special emphasis was given to the diseases of small animals and poultry during the entire meeting.

Col. Foster did a good job of stimulating interest in the A. V. M. A., and it was evident to those who heard his discussion concerning the Veterinary Corps of the U. S. Army that the profession is indeed fortunate in having had a man of his force and personality as Chief of the Veterinary Corps during the revolutionary changes that have taken place in the duties of the personnel of the Corps during the past few years. Dr. Norman J. Pyle, with his highly scientific and practical discussion, fully convinced his audience of the necessity of constant study of research work that is being done, if veterinarians are to be of real service to their clients. Dr. J. F. DeVine, of Goshen, N. Y., spoke from a wealth of experience gained from a successful practice. He stated that the time is here when veterinarians will have to specialize if they are to render the most effective service.

The small-animal clinic, conducted by Dr. Wm. J. Lentz, was scheduled for a morning session only, but there was such a wealth of material on hand that those in attendance refused to let him conclude until the close of the day. Dr. H. C. Givens reported on the progress and methods of Bang's disease eradication in Virginia. He stated that if the work could be continued at the present rate, Virginia would be practically free from this disease within the next two or three years. One of the most interesting parts of the program was a highly entertaining and scholarly lecture on "American Humor," by Dr. C. M. Newman, dean of the English Department, Virginia Polytechnic Institute. This lecture was the main feature of the banquet.

I. D. WILSON, *A. V. M. A. Res. Sec. for Virginia.*

ILLINOIS STATE VETERINARY MEDICAL ASSOCIATION

The fifty-fifth annual meeting of the Illinois State Veterinary Medical Association was held in the Abraham Lincoln Hotel, Springfield, February 18-19, 1937.

After the reading of the minutes of the previous meeting and the president's address, Dr. H. C. Rinehart, Chief Veterinarian, Springfield, discussed various state regulations. Dr. D. F. Richardson, Hoopestown, of the State Department of Agriculture, then read a well prepared paper on "Rabies Control," in which he emphasized the need for a definite program including strict licensing and annual vaccination of dogs. "Symptoms and Treatment of Cornstalk Poisoning in the Horse" was ably presented by Dr. A. J. Coale, of Carmi.

The afternoon session was opened by Dr. C. E. Dille, of Cairo, who discussed a number of unusual cases occurring in his mixed practice. Some very interesting cases were reported. A very practical paper on "Intravenous Medication" was read by Dr. E. B. Ingmand, of Zionsville, Ind. John R. Neal, M. D., chairman, Legislative Committee, Illinois State Medical Society, gave an address on "Medical Legislation," in which he pointed out the close relationship existing between the medical and veterinary professions, and how adverse medical legislation affected each group.

Mr. John P. Stout, Superintendent, Division of Animal Industry, Department of Agriculture, discussed the program for reporting animal diseases which was inaugurated in Illinois during the last year. He stated that more veterinarians are coöperating each month and making their reports promptly. The reports when compiled will be a valuable source of information. Dr. L. A. Merillat, of Chicago, brought out many interesting points in connection with the program. He called attention to several ways in which statistics covering the prevalence of communicable diseases would be of value to the live stock industry.

The dinner session was given entirely to entertainment and sociability, followed by dancing.

The morning session of February 19 was opened with an interesting discussion on "Mastitis," by Dr. H. R. Simpkins, of Elmwood. Dr. S. L. Stewart, of Olathe, Kan., read a well prepared and very practical paper on "Dairy Cattle Practice." This paper also covered many calf diseases, including white scours and pneumonia. Dr. H. E. Biester, of Iowa State College, presented a scholarly and educational lecture (illustrated) on

BANQUET, ILLINOIS STATE VETERINARY MEDICAL ASSOCIATION, SPRINGFIELD, FEBRUARY 18, 1937.

"Animal Nutrition." He brought out many valuable and practical points on the feeding of animals, which could be employed advantageously by the practitioner in his every-day work.

Dr. R. M. Hofferd, U. S. Bureau of Animal Industry, Cedar Rapids, Iowa, read a paper on "Some Phases of Swine Practice," which covered many problems encountered in swine practice. He emphasized the absolute necessity of always obtaining a complete history in every sick herd. This paper was of a very practical nature and evidenced much thought and effort in its preparation as well as a wide range of field experience.

At the afternoon session, Dr. Jesse Sampson, State Department of Agriculture, Urbana, discussed "Ketosis in Cattle and Sheep." He demonstrated the test for ketone bodies. The physiological aspect of the disease and its treatment were ably discussed and much appreciated by the members present.

"Convulsions in Dogs" was discussed by Dr. D. A. Eastman, Moline, in his usually clear, concise and practical manner. A vast amount of information covering this important subject was brought out. The discussion was greatly appreciated by the many small-animal practitioners present. A classic presentation, "The Need of an X-Ray in Small-Animal Practice and Some of Its Uses," was given by Dr. G. S. Elwood, of Chicago. In a well prepared paper, Dr. Elwood explained many uses for the x-ray and its necessity in a successful small-animal practice.

"Poultry Parasites and Nutrition" was presented by Dr. J. P. Torrey, of East Saint Louis. Dr. Torrey pointed out many cases in which parasites have interfered with proper nutrition in poultry. This talk brought out a vast amount of information that was highly valued by all present.

At the business session, 45 applications for membership were received and accepted.

Officers elected for the coming year were: President, Dr. G. W. Jensen, Antioch; vice-president, Dr. C. E. Dille, Cairo; secretary-treasurer, Dr. C. C. Hastings (reëlected), Williamsville, and member of Executive Board, Dr. C. L. Miller, Oak Park.

C. C. HASTINGS, *Secretary-Treasurer.*

Up-to-Date

Proud Dairy Farmer: "Yep, we've got all the new-fangled machinery there is. Our latest is two milking machines."

City Miss: "How interesting. But tell me, do they really give as good milk as cows?"

Goat World.

NECROLOGY

PETER BENJAMIN BROWN

Dr. Peter B. Brown, of Meriden, Conn., died at his home, December 29, 1936, as a result of a heart attack. He had been feeling well a few hours prior to his death, having made several professional calls in the morning.

Born at Middletown, Conn., July 20, 1897, Dr. Brown attended the Havlin (N. Y.) High School and studied veterinary medicine at the University of Pennsylvania, from which he was graduated with the class of 1916. Following graduation, he engaged in general practice in Meriden and remained there until death overtook him. Dr. Brown served his city as inspector of meat and other foods.

Dr. Brown joined the A. V. M. A. in 1916. He was a member of Alpha Psi Fraternity, president of the Meriden Grange, Inc., and a member of the Lulu Temple Shrine, in Philadelphia. He is survived by his widow (née Elizabeth A. Kirshner), one sister and one brother. A. H. C.

RALPH ALEXANDER DUNN

Dr. Ralph A. Dunn, of Santa Ana, Calif., died January 16, 1937, of heart failure.

Born November 28, 1870, at Jamestown, N. Y., Dr. Dunn attended local schools and the American Veterinary College, New York City. Following his graduation in 1891, he practiced in Alexandria, Va., where he also was veterinarian to the local Board of Health. Later he removed to Charlottesville, Va., where he practiced until 1929, when he went to California. He was in Beverly Hills for about a year, before going to Santa Ana.

During the World War, Dr. Dunn served in the Veterinary Corps of the Regular Army. He was commissioned as a second lieutenant, August 24, 1917, and was discharged on October 2, 1920, with the rank of major. During his period of service he was stationed at the Port of Embarkation, Newport News, Va., and at Camp Dix, N. J., as Camp Veterinarian.

Dr. Dunn joined the A. V. M. A. in 1929. He was a member of the Twelfth International Veterinary Congress.

CHARLES GOFF THOMSON

Colonel Charles G. Thomson, superintendent of Yosemite National Park, died at Lewis Memorial Hospital, Yosemite, Calif., on March 23, 1937, after an illness of three days.

Born at Little Falls, N. Y., February 9, 1883, Col. Thomson studied veterinary medicine at Cornell University and was graduated in 1907. The same year he went to the Philippines to succeed the late Dr. A. S. Shealy (Iowa '03) at the Antirinderpest Serum Laboratory, at San Lazaro. At that time, Dr. Victor Buencamino, now Chief of the Philippine Bureau of Animal Industry, was an assistant in the laboratory, later going to Cornell to study veterinary medicine. In 1912, Dr. Thomson was in charge of the Alabang Stock Farm, where the serum laboratories were located, with Dr. W. H. Boynton (Corn. '08) then in charge. Later Dr. Thomson was assistant director of prisons for the Philippine Government. He was a member of the A. V. M. A. from 1910 until 1916.

At the outbreak of the World War, Thomson was placed in charge of the Remount Division, commanding remount depots at Camp Gordon and Camp Dix. In July, 1918, he was promoted to major and sent to France. A later advancement to the rank of colonel put him in command of all troops at Lux, France, until May, 1919. He received a citation for "exceptionally meritorious and conspicuous services" during the war.

Colonel Thomson wrote several books, among them "Terry —a Tale of the Hill People." Last year, he was loaned to the Mexican Government to assist that country in organizing and administering a system of national parks.

SIDNEY E. ALLISON

Dr. Sidney E. Allison, of Benton, Ky., died at his home, March 24, 1937, after an illness of about two weeks, caused by an attack of influenza.

Born in Lyon County, Ky., January 25, 1885, Dr. Allison was graduated from the Indiana Veterinary College in 1917. He practiced first at Birmingham, going to Benton about ten years ago. He is survived by his widow (née Audra Gibson), two sisters and two brothers.

J. HOUSTON WISE

Dr. J. Houston Wise, of Deer Lick, Pa., died on January 18, 1937, at the age of 82 years. He was a non-graduate who registered under the Act of 1905.

HUB H. GUY

Dr. Hub H. Guy, of Petersburg, Mich., died March 15, 1937. He was a graduate of the Grand Rapids Veterinary College, class of 1910, and practiced at Walworth, Mich., before going to Petersburg.

J. D. EBRIGHT

Dr. J. D. Ebright, of Gary, Ind., died in Mercy Hospital, February 15, 1937, following a lingering illness. He had suffered a stroke of paralysis two years previously.

Born in Shreve, Ohio, June 17, 1859, Dr. Ebright attended the Ontario Veterinary College. He entered practice at Crown Point, Ind., later going to Hammond. He had been practicing in Gary since 1913. Surviving him are a daughter and a son, Dr. Glenn L. Ebright (Chi. '08), of Hammond.

WILLIAM AYLMER THOMPSON

Dr. William A. Thompson, of Rushville, N. Y., died at his home on March 5, 1937, following an apoplectic stroke.

Born at Holt, Ontario, Canada, August 22, 1881, Dr. Thompson attended local schools and the Ontario Veterinary College. Following his graduation in 1911, he practiced at Elmvale, Ont. He removed to Rushville, N. Y., about four years ago.

Dr. Thompson joined the A. V. M. A. in 1922. He is survived by his widow, two sons and one daughter.

VICTOR MARIS MONTGOMERY

Dr. Victor M. Montgomery, of Elnora, Ind., died in the Linton (Ind.) Hospital, March 9, 1937, of rabies. Eight weeks previously, he had been bitten on the hand by a dog and had started to take the Pasteur treatment. For some reason this was stopped after Dr. Montgomery had received 14 injections. Five days before his death he became violent and was removed to the hospital.

Born in Vallene, Ind., in 1886, Dr. Montgomery studied veterinary medicine first at the Chicago Veterinary College and later at the Indiana Veterinary College. He held diplomas from both institutions, received in 1909 and 1921, respectively. He practiced at Jasper, Ind., before locating at Elnora, four years ago. He is survived by his widow (née Hazel Robinson), two stepchildren and three sisters.

Dr. Montgomery was a World War veteran.

GUSTAVE G. RATZ

Dr. Gustave G. Ratz, of Red Bud, Ill., was found dead in his home, March 24, 1937. He had been dead about six hours, death having followed a cerebral hemorrhage.

Born in Red Bud, March 22, 1873, Dr. Ratz was a graduate of the Ontario Veterinary college, class of 1895, and had practiced in Red Bud over 40 years. He had been president of the local School Board and treasurer of the volunteer fire department at Red Bud for the past 30 years.

Dr. Ratz was a member of the Illinois State Veterinary Medical Association.

BERNARD M. COLLINS

Dr. Bernard M. Collins, of Pittsfield, Mass., died March 26, 1937, at the House of Mercy, where he had been a patient since February 3.

Born in Pittsfield, January 17, 1889, Dr. Collins was graduated from the Pittsfield High School and then entered the University of Pennsylvania, where he was graduated from the Veterinary School in 1911. He entered practice and served for a time as a state meat inspector. He organized the Yellow Cab Company and was its president until he retired in 1925. Later he maintained a pet shop in Pittsfield and, in 1930, established kennels on South Street, Pittsfield. He specialized in Scottish terriers. He was in demand among dog lovers and frequently was invited to act in a professional capacity at horse and dog shows. At the time of his death, he was proprietor of the Berkshire Hills Kennels.

Dr. Collins is survived by his widow (née Elizabeth A. Murphy), three sons, six daughters, his parents, three brothers and three sisters.

E. L. S.

OTIS BARNETT

Dr. Otis Barnett, of Edwardsville, Ill., died March 29, 1937, at the age of 72, in Saint Elizabeth's Hospital at Granite City, Ill., following a paralytic stroke which he suffered the day before at his home. Dr. Barnett was a graduate of the Chicago Veterinary College, class of 1891, and practiced at Edwardsville for 35 years. He retired from practice about ten years ago. He was a member of the A. V. M. A. from 1892 to 1896.

WILLIAM L. HIATT

Dr. William L. Hiatt died at his home in Edmond, Okla., March 26, 1937, of angina pectoris. He had been ill and confined to his bed about three weeks prior to his death.

Born in Westland, Ind., January 19, 1873, Dr. Hiatt spent his boyhood days there. In 1884, his parents moved to Kansas. He entered the Ontario Veterinary College in 1898, transferred to the McKillip Veterinary College and later the Chicago Veterinary College, and was graduated from the latter in 1902. Immediately upon his graduation, he entered the service of the B. A. I., receiving an assignment to meat inspection in Wichita, Kan. In 1905, he was transferred to Oklahoma on tick eradication work. With the exception of a short period spent in Kansas City on meat inspection, he was engaged in various Bureau field projects, and remained in Oklahoma until his death.

Dr. Hiatt joined the A. V. M. A. in 1929. He was a charter member of the Oklahoma Veterinary Medical Association, serving as its President during 1936. He was also a member of the National Association of B. A. I. Veterinarians and the Twelfth International Veterinary Congress. Mourning his death are his widow (née Myra Pearl Zimmerman), four sons and four daughters.

C. H. F.

WILLIAM DENT MASON

Dr. William D. Mason, of Elko, Nev., ended his life with a self-inflicted bullet wound, at his home, April 2, 1937. No reason could be found for his rash act.

Born in Winchester, Ohio, in June, 1875, Dr. Mason started the study of veterinary medicine at Iowa State College. He completed his studies at Washington State College in 1906. He entered the service of the U. S. Bureau of Animal Industry and, in 1912, when stationed in Elko, was transferred to Alabama. However, rather than leave Elko, he resigned from the service and organized the Mason Products Company, a milk distributing concern. This proved to be a very successful enterprise.

Dr. Mason joined the A. V. M. A. in 1915. He was regarded as one of the outstanding citizens of Elko and was always interested in any civic undertaking that would promote the interests of his city. He was particularly interested in horse racing and football. He was an Elk and a Mason. He is survived by his widow, his mother, one brother and three sisters.

E. R.

EZRA STRICKLAND DEUBLER

Dr. Ezra S. Deubler died at his home, Penshurst Farm, Narberth, Pa., March 30, 1937, after an illness of about four months.

Born April 20, 1879, Dr. Deubler was a graduate of the University of Pennsylvania, class of 1905. He immediately entered the service of the Pennsylvania State Live Stock Sanitary Board under the late Dr. Leonard Pearson. Dr. Deubler was placed in charge of the experiment farm of the Board, near Media, Pa., a project designed by Dr. Pearson for studying infectious diseases of animals under actual farm conditions. Special attention was given to bovine tuberculosis, Bang's disease, hog cholera, epizoötic lymphangitis and Johne's disease. Dr. Deubler continued in this work under the late Dr. S. H. Gilliland and then Dr. C. J. Marshall, state veterinarians of Pennsylvania following Dr. Pearson.

In 1912, Dr. Deubler left state employ to become manager of Penshurst Farm, owned by Mr. Percival Roberts. Here Dr. Deubler played a prominent rôle in founding and developing the now famous Penshurst Ayrshires. He served as president of both the state and national Ayrshire associations, as president of the Pennsylvania Dairymen's Association, as president of the Bala-Cynwyd-Narberth Rotary Club, and as a member of the Lower Merion School Board.

Dr. Deubler joined the A. V. M. A. in 1906. He was a member of the Twelfth International Veterinary Congress. In 1935 he was awarded a distinguished service medal by the Philadelphia Society for Promoting Agriculture, for his outstanding work in the fields of veterinary science and dairy husbandry. He is survived by his widow and four children.

JAMES VERNE JONES

Dr. J. Verne Jones, of Cheviot, Ohio, died at his home, April 7, 1937, following a heart attack. He had suffered from a cardiac condition for several years and had been confined to his home for several weeks.

Born at Okeana, Ohio, April 9, 1892, Dr. Jones attended local schools and the Indiana Veterinary College. He was graduated in 1920, and from 1921 to 1930 served as chief veterinarian for the Hamilton County (Ohio) Board of Health. Since that time he had been in private practice.

Dr. Jones joined the A. V. M. A. in 1936. He was a member of the Scottish Rite, Shrine, Snow Lodge, F. & A. M., and the Elks. He is survived by his widow (née Eunice Kendrick).

REX A. WHITING

Dr. Rex A. Whiting, of San Diego, Calif., died at his home on March 27, 1937, from heart disease.

Born in 1884, Dr. Whiting was graduated from the New York State Veterinary College at Cornell University in 1905. The following year he took postgraduate work at his Alma Mater and later special work at the University of Pennsylvania and the University of Michigan. From 1908 to 1910, he was in the meat inspection service of the U. S. Bureau of Animal Industry. In 1911, he went to Purdue University as associate veterinarian under Dr. R. A. Craig and remained in that position for almost

DR. R. A. WHITING

18 years. In 1928, he went to San Diego, Calif., as pathologist and director of research for the Zoölogical Society of San Diego, at Balboa Park. In 1932, he resigned to enter private practice in San Diego. He conducted a diagnostic laboratory in connection with his practice. He also served as veterinarian to the San Diego County Health Department.

Dr. Whiting joined the A. V. M. A. in 1916. He was a member of the Twelfth International Veterinary Congress, Lafayette (Ind.) Lodge No. 123 F. & A. M., Lafayette Commandery No. 3, Knights Templar and Zorah Temple A. A. O. N. M. S. of Terre Haute, Ind. He is survived by his widow and two daughters.

HARRY JAMES ELLIOTT

Dr. Harry J. Elliott, of Brandon, Man., passed away at Winnipeg, Man., on April 2, 1937.

Born at Port Hope, Ont., August 18, 1874, Dr. Elliott moved with his parents to Wawanesa, Man., in the early 80's. He attended the Ontario Veterinary College but finished his veterinary education at the McKillip Veterinary College, in 1900. He started practice at Brandon, where he remained for a few years. Then he entered the Department of Indian Affairs, at Ottawa. Later he was appointed to the Health of Animals Branch, Department of Agriculture, where he served until the outbreak of the war in 1914. He went overseas and served until the close of the war, in 1919. He attained the rank of lieutenant colonel. He resumed his work with the Health of Animals Branch and served at Pinhorn, Alta., and North Portal, Sask. In 1935, he was superannuated owing to war disability.

Dr. Elliott is survived by his widow (née Helena C. Quinn), two sisters and three brothers.

A. E. C.

ABRAHAM G. COX

Dr. Abraham G. Cox, of Carlisle, Ind., died at his home, April 4, 1937, at the age of 79 years.

Born in Haddon Township, Sullivan County, Ind., in 1858, Dr. Cox attended the Ontario Veterinary College and the Indiana Veterinary College. He was graduated from the latter in 1898 and located in Carlisle, where he practiced until his death.

Dr. Cox joined the A. V. M. A. in 1912. He was a trustee of Haddon Township from 1901 until 1903. He is survived by his widow, one daughter, two sisters and two brothers.

ANDREW J. ABBOTT

Dr. Andrew J. Abbott, of Marshfield, Wis., died at his home, April 7, 1937. Heart failure was the cause of death, which followed an illness of about a week.

Born at Whitehall, Wis., February 10, 1877, Dr. Abbott attended local grade and high schools and then entered the Ontario Veterinary College. Following his graduation in 1907, he located in Marshfield and practiced there until his death. He was prominent in civic affairs and for four years served his ward as alderman on the Common Council. When his term expired in 1932,

he declined to be a candidate again. During his tenure as alderman, Dr. Abbott served as chairman of the Board of Health. Fraternally he was identified with the Masons, Odd Fellows, Elks, Woodmen and the Equitable Fraternal Union.

Dr. Abbott joined the A. V. M. A. in 1913. He was a member of the Twelfth International Veterinary Congress, the Wisconsin State Veterinary Medical Association and the Central Wisconsin Veterinary Society. He was vice-president of the Wisconsin State Board of Veterinary Examiners for a number of years. He is survived by one son, an adopted daughter, four brothers and one sister.

J. S. H.

SIMON P. SMITH

Dr. S. P. Smith, of Washburn, Ill., died at his home, March 31, 1937, following an illness of two weeks. He was a graduate of the McKillip Veterinary College, class of 1900, and had practiced at Washburn for about 16 years. Prior to locating in Washburn, he practiced at Martinsville and Altamont, Ill. He is survived by his widow, two daughters, three sons, two sisters and two brothers.

ROBERT E. WASHBURN

Dr. Robert E. Washburn, of Gloversville, N. Y., died in the Nathan Littauer Hospital, April 13, 1937. He had been critically ill for a week. He suffered a heart attack two weeks before his death and complications developed.

Born in Benson Center, N. Y., August 18, 1885, Dr. Washburn received his early education in the local schools and then entered the McKillip Veterinary College. Following his graduation in 1909, he located in Havana, Cuba, where he conducted a hospital and practice in partnership with a classmate.

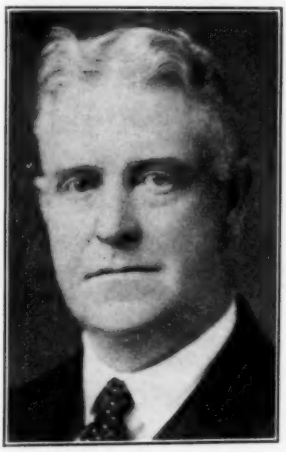
At the outbreak of the World War, Dr. Washburn enlisted as a private and rose to the rank of captain in the Veterinary Corps. He reported at the Veterinary Training School at Camp Lee, Va., August 2, 1918. The same month he was assigned to Mobile Veterinary Hospital No. 3. This unit was sent to Coblenz, Germany, with the Third Army. He was discharged on August 30, 1919, and, upon returning to this country entered practice again, in partnership with the late Dr. Henry S. Cady. Later Dr. Washburn acquired Dr. Cady's interest.

Dr. Washburn joined the A. V. M. A. in 1927. He was a member of the New York State Veterinary Medical Society, the

Hudson Valley Veterinary Society, the Twelfth International Veterinary Congress, the New York State Association of Dairy and Milk Inspectors, the Association of Military Surgeons of the United States, the American Legion, the Veterans of Foreign Wars and a number of other military organizations. The funeral, marked with solemn and impressive military and religious ceremonies, was one of the largest ever held in Gloversville. He is survived by his widow, two sisters and one brother.

NICHOLAS F. WILLIAMS

Death has taken another past president of the American Veterinary Medical Association—Dr. N. F. Williams, of Philadelphia, Pa. He had been ill for several weeks and had undergone an operation for intestinal carcinoma. He died on April 8, 1937, and funeral services were held in Boston, Mass., on the 12th.



DR. N. F. WILLIAMS

Born in Boston, July 27, 1872, Dr. Williams was graduated from the Kansas City Veterinary College in 1911. He entered practice at Amarillo, Texas, and remained there for about 15 years. In 1926, "Ma" Ferguson, then governor of Texas, appointed Dr. Williams to the office of State Veterinarian. He also served as a member and as president of the Texas State Veterinary Medical Examining Board.

Dr. Williams joined the A. V. M. A. in 1917. He served as secretary of the Section on Sanitary Science and Food Hygiene for two terms (1925-27) and as a member of the Committee on

Resolutions, 1926-27. He served as Resident Secretary for Texas, 1928-30, and was elected a member of the Executive Board in 1930, for a one-year term, and the following year was elected for a five-year term, representing District 8 (Kansas, Oklahoma, Missouri, Arkansas, Texas and Louisiana). While a member of the Board he was elected to the presidency of the A. V. M. A. This was at the meeting in Atlanta in 1932. During his term as president, Dr. Williams visited ten states in the interests of the A. V. M. A. Dr. Williams also was a member of the Twelfth International Veterinary Congress.

On August 8, 1933, Dr. Williams joined the Jensen-Salsbery Laboratories as manager of the Philadelphia depot of the organization. Because of his wonderful personality and splendid executive ability, he was very successful in this new undertaking. He had a very deep interest in the veterinary profession and was a staunch supporter of organized veterinary medicine on all occasions. He was a natural orator and was never found wanting when called upon to speak at gatherings of veterinarians or stockmen.

PERSONALS

MARRIAGES

Dr. W. H. Lindley (K. S. C. '33), to Miss Olga Mae Jarvis, both of Lawrenceburg, Ind., at Covington, Ky., February 20, 1937.

Dr. J. La Vere Davidson (O. S. U. '33), of Kalamazoo, Mich., to Miss Anne Lawrence, of Vicksburg, Mich., at Lagrange, Ind., March 20, 1937.

PERSONALS

Dr. J. F. Robb (Ont. '32), has removed from Saline, Mich., to Jackson, same state.

Dr. W. J. Hadder (Gr. Rap. '09), of Mecosta, Mich., was on the sick list most of the winter.

Dr. W. A. Young (N. Y.-Amer. '00), for many years located at Boonville, N. Y., is now at Wausau, Wis.

Dr. H. G. Hock (Ind. '21), who practiced in Andover, Ohio, for twelve years, has removed to Hubbard, Ohio.

Dr. W. L. Derrer (Chi. '20), of Sterling, Ill., recently completed extensive alterations in his veterinary hospital.

Dr. Hobert Fairfield (McK. '10) has resumed practice at Monticello, Ind., after spending several years in other pursuits.

Dr. Richard A. Huebner (U. P. '36) reports a change of address from Coatesville, Pa., to 2723 North 13th Street, Philadelphia, Pa.

Dr. John P. Turner (U. P. '90), of Washington, D. C., was confined to his home on account of illness for two weeks during April.

DR. JOHN W. TRUMBLE (Mich. '34), of Lowell, Mich., has received a permanent appointment in the U. S. Bureau of Animal Industry.

DR. WALTER J. WILLIAMS (Corn. '36) has requested a change of address from Lexington, Ky., to 80 North Broadway, White Plains, N. Y.

DR. E. ZIEGENBUSCH (Cin. '12), has returned to Delphos, Ohio. Accompanied by Mrs. Ziegenbusch, he spent about four months in Miami, Fla.

DR. A. S. WILLARD (Ind. '22), of Edinburg, Ind., has purchased a building which he is remodeling for occupancy as a residence and office.

DR. J. R. BROWN (O. S. U. '20), of Ottawa, Ill., was a hospital patient the latter part of March. He had his gall-bladder and his appendix removed.

DR. FLOYD VOLLMER (O. S. U. '36), who has been in the service of the federal government recently, has resigned to reënter practice at Liberty, Ill.

DR. THEODORE W. BROWN (A. P. I. '35) has resigned his position with the U. S. Bureau of Animal Industry and has entered practice at Greenville, Tenn.

DR. BEN ANDERSON (Iowa '26), of Canton, S. Dak., has been appointed Director of the Division of Animal Industry, South Dakota Department of Agriculture.

DR. WILLIAM R. ROSEKRANS (Amer. '96), of the U. S. B. A. I. meat inspection force at New York City, was retired from active service, February 28, 1937.

DR. R. H. FISH (Mich. '31), of Albion, Mich., has accepted an appointment in the U. S. Bureau of Animal Industry and is stationed at East Lansing, Mich.

DR. W. P. TAGUE (Ind. '22), of Chicago, Ill., has purchased a farm, a mile east of Antioch, Ill., where he plans to operate a breeding and boarding kennel.

DR. S. S. MORGAN (K. C. V. C. '12), formerly of Pensacola, Fla., has accepted a position as Supervisor of the Food and Sanitary Department, City of Gainesville, Fla.

DR. A. B. PLETSCH (T. H. '12), of McNabb, Ill., was elected supervisor of Magnolia Township, Putnam County, on the Republican ticket, at the election held April 6.

DR. WM. HENRY KELLY (N. Y. C. V. S. '89), of Albany, N. Y., has returned home after spending several weeks basking in the Florida sunshine in the interests of his health.

DR. E. C. GOODRICH (Gr. Rap. '15), of Kalamazoo, Mich., has found it necessary to discontinue active practice on account of poor health. He went to Louisville, Ky., in April.

DR. E. B. ACKERMAN (Amer. '91), of Huntington, L. I., N. Y., was operated upon for a strangulated hernia recently and has gone to Florida to recuperate from the operation.

DR. ANDREW ENGLISH (Corn. '05), of Omaha, Neb., was retired from the service of the U. S. Bureau of Animal Industry, February 28, 1937. He was on the meat inspection force.

DR. L. F. CONTI (San Fran. '17), has severed his connection with the Los Angeles County Live Stock Department to accept a position with the Zoölogical Society of San Diego.

Dr. G. DONALD PINDER (Ont. '34), has about completed a new hospital for small animals at 1195 W. Broadway, Vancouver, B. C. It will be known as the Alderway Pet Hospital.

DR. A. MAURICE STARK (Corn. '10), of New York City, is building a new hospital at 144 East 57th St., which was the subject of a feature article in one of the New York newspapers recently.

DR. J. T. BURRISS (O. S. U. '31) is now affiliated with the National Laboratories Corporation as Ohio field representative. He will make his headquarters at 431 Larcomb Avenue, Columbus, Ohio.

DR. ARTHUR SELIGMAN (N. Y. U. '20), of Brooklyn, N. Y., has entered the service of the U. S. Bureau of Animal Industry and is stationed at Knoxville, Tenn., on Bang's disease eradication work.

DR. JOHN L. WRIGHT (U. P. '32) has resigned his position with the U. S. Bureau of Animal Industry and has returned to private practice at Lagrange, Ga., where he has a hospital for small animals.

DR. LEO DUNLAP (O. S. U. '33), of Zanesville, Ohio, has taken a long-time lease on a building located at 62 South Sixth Street. After some alterations are made, he will occupy it as a veterinary hospital.

DR. F. A. ZIMMER (O. S. U. '09), State Veterinarian of Ohio, addressed the Northern Ohio Draft Horse Association, at a meeting held in Wellington, Ohio, April 6. His subject was "Diseases of the Horse."

DR. J. P. FOSTER (Ont. '00—McK. '08—Ont. '14), of Minneapolis, Minn., addressed the Minneapolis Saddle and Bridle Club at a recent noonday luncheon. His remarks covered the comparative anatomy of the horse and man.

DR. G. M. FUNKHOUSER (McK. '08), of Lafayette, Ind., has been appointed City Milk Inspector by Mayor Teal. Dr. Funkhouser held the same position under two former mayors, from 1914 to 1918, and from 1926 to 1930.

DR. A. A. ROHRER (O. S. U. '32), of Columbus, Ohio, recently completed the construction of a small-animal hospital in conjunction with a home. The building follows the style of veterinary hospitals built on the Pacific Coast.

DR. W. A. HAHN (Chi. '16), of Geneva, Ill., sustained painful injuries in an automobile accident on the Lincoln Highway seven miles west of Geneva, the latter part of March. A cattle truck crashed into the rear of Dr. Hahn's coupé.

DR. MAURICE C. HALL (Geo. Wash. '16), of Washington, D. C., was a patient in Walter Reed Hospital, in April. His stomach had been giving him some trouble again, but the most recent report was to the effect that "the Army had the situation well in hand."

DR. O. NORLING-CHRISTENSEN (O. S. U. '32), of Chicago, Ill., has purchased the Youngman Kennels and seven acres of ground on Hibbard Road in Wilmette, Ill. He will reside on the premises and operate this unit as one of the Christensen Animal Hospitals.

DR. O. H. MUTH (Mich. '29) has resigned his position with the Wisconsin Department of Farms and Markets and has returned to the Oregon State Agricultural College as assistant professor of veterinary medicine, to engage in investigational work with diseases of range animals.

DR. CHAS. R. SCHROEDER (Wash. '29), who has been pathologist to the Zoölogical Society of San Diego, at Balboa Park, for several years, recently resigned to accept a similar position at the New York Zoölogical Park. Dr. Schroeder fills the vacancy caused by the death of Dr. Charles V. Noback.

DR. C. A. BRADLEY (Chi. '97), of Marion, Iowa, has been getting considerable pleasure out of breeding "midget mules," by crossing a very small jack on Shetland pony mares. The offspring show all of the characteristics of the ordinary mule, are very nice to handle, learn easily and take to their work quite readily, according to Dr. Bradley. The total weight, at birth, of five midget mules was 151 pounds. Two of them, "Esau" and "Rachel," have been fitted with silver shoes.